Doc Code: PET.OP

Document Description: Petition for Review by the Office of Petitions

IAP03Rec'd PCT 27 AUG 2009

PTO/SB/64 (04-09)
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PETITION FOR REVIVAL OF AN APPLICATION FOR PATENT ABANDONED UNINTENTIONALLY UNDER 37 CFR 1.137(b)	Docket Number (Optional) 8114-008WO-US

First named inventor: Adrian I. Bot

Application No.: 10/527,931 Art Unit: 1633

Filed: August 26, 2005 Examiner: WEHBE, ANNE MARIE SABRINA

Title: Methods and compositions to generate and control the effector profile of t cells by simultaneous loading and activation of selected subsets of antigen presenting cell

Attention: Office of Petitions
Mail Stop Petition
Commissioner for Patents
P.O. Box 1450
Alexandria, VA 22313-1450
FAX (571) 273-8300

NOTE: If information or assistance is needed in completing this form, please contact Petitions Information at (571) 272-3282.

The above-identified application became abandoned for failure to file a timely and proper reply to a notice or action by the United States Patent and Trademark Office. The date of abandonment is the day after the expiration date of the period set for reply in the office notice or action plus any extensions of time actually obtained.

APPLICANT HEREBY PETITIONS FOR REVIVAL OF THIS APPLICATION

NOTE: A grantable petition requires the following items:
(1) Petition fee;
(2) Reply and/or issue fee;
(3) Terminal disclaimer with disclaimer fee - required for all utility and plant applications filed before June 8, 1995; and for all design applications; and
(4) Statement that the entire delay was unintentional

1. Petition Fee

☑ Small entity-fee $ 810.00 (37 CFR 1.17(m)). Application claims small entity status. See 37 CFR 1.27.

☐ Other than small entity-fee $ _____ (37 CFR 1.17(m))

2. Reply and/or fee

A. The reply and/or fee to the above-noted Office action in the form of a response to the Office Action (identify type of reply):

☐ has been filed previously on _____.

☑ is enclosed herewith.

08/28/2009 CCHAU1 00000024 10527931
01 FC:2453 810.00 OP

B. The issue fee and publication fee (if applicable) of $ _____.

☐ has been paid previously on _____.

☐ is enclosed herewith.

If you need assistance in completing the form, call 1-800-PTO-9199 and select option 2.

3. Terminal disclaimer with disclaimer fee

☑ Since this utility/plant application was filed on or after June 8, 1995, no terminal disclaimer is required.

☐ A terminal disclaimer (and disclaimer fee (37 CFR 1.20(d)) of $_____for a small entity or $_____ for other than a small entity) disclaiming the required period of time is enclosed herewith (see PTO/SB/63).

4. STATEMENT: The entire delay in filing the required reply from the due date for the required reply until the filing of a grantable petition under 37 CFR 1.137(b) was unintentional. [NOTE: The United States Patent and Trademark Office may require additional information if there is a question as to whether either the abandonment or the delay in filing a petition under 37 CFR 1.137(b) was unintentional (MPEP 711.03(c), subsections (III)(C) and (D)).]

WARNING:

Petitioner/applicant is cautioned to avoid submitting personal information in documents filed in a patent application that may contribute to identity theft. Personal information such as social security numbers, bank account numbers, or credit card numbers (other than a check or credit card authorization form PTO-2038 submitted for payment purposes) is never required by the USPTO to support a petition or an application. If this type of personal information is included in documents submitted to the USPTO, petitioners/applicants should consider redacting such personal information from the documents before submitting them to the USPTO. Petitioner/applicant is advised that the record of a patent application is available to the public after publication of the application (unless a non-publication request in compliance with 37 CFR 1.213(a) is made in the application) or issuance of a patent. Furthermore, the record from an abandoned application may also be available to the public if the application is referenced in a published application or an issued patent (see 37 CFR 1.14). Checks and credit card authorization forms PTO-2038 submitted for payment purposes are not retained in the application file and therefore are not publicly available.

David Lewis
Signature

Aug 24, 2009
Date

David Lewis
Type or Printed name

33,101
Registration Number, If applicable

1250 Aviation Avenue, Suite 200B
Address

(408) 993-1800
Telephone Number

San Jose, CA 95110
Address

Enclosures:

☑ Fee Payment

☑ Reply

☐ Terminal Disclaimer Form

☑ Additional sheets containing statements establishing unintentional delay

☐ Other: _____

CERTIFICATE OF MAILING OR TRANSMISSION [37 CFR 1.8(a)]

I hereby certify that this correspondence is being:

☑ Deposited with the United States Postal Service on the date shown below with sufficient postage as first class mail in an envelope addressed to: Mail Stop Petition, Commissioner for Patents, P. O. Box 1450, Alexandria, VA 22313-1450.

☐ Transmitted by facsimile on the date shown below to the United States Patent and Trademark Office at (571) 273-8300.

Aug 24, 2009
Date

David Lewis
Signature

David Lewis
Typed or printed name of person signing certificate

Doc Code: PET.OP

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PTO/SB/64 (04-09)
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PETITION FOR REVIVAL OF AN APPLICATION FOR PATENT ABANDONED UNINTENTIONALLY UNDER 37 CFR 1.137(b)

Docket Number (Optional): 8114-008WO-US

First named inventor: Adrian I. Bot

Application No.: 10/527,931

Art Unit: 1633

Filed: August 26, 2005

Examiner: WEHBE, ANNE MARIE SABRINA

Title: Methods and compositions to generate and control the effector profile of t cells by simultaneous loading and activation of selected subsets of antigen presenting cell

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(2) Reply and/or issue fee;
(3) Terminal disclaimer with disclaimer fee - required for all utility and plant applications filed before June 8, 1995; and for all design applications; and
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☐ Other than small entity-fee $ _____ (37 CFR 1.17(m))

2. Reply and/or fee

A. The reply and/or fee to the above-noted Office action in the form of a response to the Office Action (identify type of reply):

☐ has been filed previously on _____.

☑ is enclosed herewith.

B. The issue fee and publication fee (if applicable) of $_____.

☐ has been paid previously on _____.

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David Lewis
Signature

Aug 24, 2009
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David Lewis
Type or Printed name

33,101
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San Jose, CA 95110
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☐ Other: _____

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Aug 24, 2009
Date

David Lewis
Signature

David Lewis
Typed or printed name of person signing certificate

PTO/SB/21 (04-07)
Approved for use through 09/30/2007. OMB 0651-0031
U.S. Patent and Trademark Office; U.S. DEPARTMENT OF COMMERCE

TRANSMITTAL FORM

(to be used for all correspondence after initial filing)

Total Number of Pages in This Submission: 134

Application Number	10/527,931
Filing Date	August 26, 2005
First Named Inventor	Adrian I. Bot
Art Unit	1633
Examiner Name	WEHBE, ANNE MARIE SABRINA
Attorney Docket Number	8114-008-WO-US

ENCLOSURES *(Check all that apply)*

- [x] Fee Transmittal Form
- [x] Fee Attached
- [x] Amendment/Reply
- [ ] After Final
- [x] Affidavits/declaration(s)
- [ ] Extension of Time Request
- [ ] Express Abandonment Request
- [ ] Information Disclosure Statement
- [ ] Certified Copy of Priority Document(s)
- [ ] Reply to Missing Parts/ Incomplete Application
- [ ] Reply to Missing Parts under 37 CFR 1.52 or 1.53
- [ ] Drawing(s)
- [ ] Licensing-related Papers
- [x] Petition
- [ ] Petition to Convert to a Provisional Application
- [ ] Power of Attorney, Revocation Change of Correspondence Address
- [ ] Terminal Disclaimer
- [ ] Request for Refund
- [x] CD, Number of CD(s) 2
- [ ] Landscape Table on CD
- [ ] After Allowance Communication to TC
- [ ] Appeal Communication to Board of Appeals and Interferences
- [ ] Appeal Communication to TC **(Appeal Notice, Brief, Reply Brief)**
- [ ] Proprietary Information
- [ ] Status Letter
- [x] Other Enclosure(s) (please Identify below):

Return Receipt Postcard, Check for $914, Sequence Listing, and Petition to revive / explanation which includes: Exhibit I - Emails and Letters, Exhibit II - Affidavit of W. Gerald Newmin, Exhibit III - Affidavit of Martin Schroeder, Exhibit IV - Affidavit of David Lewis

Remarks

The Return Receipt Postcard, the check, and the exhibit cover sheets are not included in the total number of pages.

SIGNATURE OF APPLICANT, ATTORNEY, OR AGENT

Firm Name	David Lewis, Registered Patent Agent		
Signature	Dave Lewis		
Printed name	David Lewis		
Date	Aug 24, 2009	Reg. No.	33,101

CERTIFICATE OF TRANSMISSION/MAILING

I hereby certify that this correspondence is being facsimile transmitted to the USPTO or deposited with the United States Postal Service with sufficient postage as first class mail in an envelope addressed to: Commissioner for Patents, P.O. Box 1450, Alexandria, VA 22313-1450 on the date shown below:

Signature	Dave Lewis		
Typed or printed name	David Lewis	Date	Aug 24, 2009

PTO/SB/17 (10-08)
Approved for use through 06/30/2010. OMB 0651-0032
U.S. Patent and Trademark Office; U.S. DEPARTMENT OF COMMERCE
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Effective on 12/08/2004.
Fees Pursuant to the Consolidated Appropriations Act, 2005 (H.R. 4818).

FEE TRANSMITTAL
For FY 2009

☑ Applicant claims small entity status. See 37 CFR 1.27

TOTAL AMOUNT OF PAYMENT	($)	914

Complete if Known	
Application Number	10/527,931
Filing Date	August 26, 2005
First Named Inventor	Adrian I. Bot
Examiner Name	WEHBE, ANNE MARIE SABRINA
Art Unit	1633
Attorney Docket No.	8114-008-WO-US

METHOD OF PAYMENT (check all that apply)

☑ Check ☐ Credit Card ☐ Money Order ☐ None ☐ Other (please identify): _____

☑ Deposit Account Deposit Account Number: 503345 Deposit Account Name: _____

For the above-identified deposit account, the Director is hereby authorized to: (check all that apply)

☐ Charge fee(s) indicated below ☐ Charge fee(s) indicated below, **except for the filing fee**

☑ Charge any additional fee(s) or underpayments of fee(s) under 37 CFR 1.16 and 1.17 ☑ Credit any overpayments

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FEE CALCULATION

1. BASIC FILING, SEARCH, AND EXAMINATION FEES

Application Type	FILING FEES Fee ($)	FILING FEES Small Entity Fee ($)	SEARCH FEES Fee ($)	SEARCH FEES Small Entity Fee ($)	EXAMINATION FEES Fee ($)	EXAMINATION FEES Small Entity Fee ($)	Fees Paid ($)
Utility	330	165	540	270	220	110	
Design	220	110	100	50	140	70	
Plant	220	110	330	165	170	85	
Reissue	330	165	540	270	650	325	
Provisional	220	110	0	0	0	0	

2. EXCESS CLAIM FEES

Fee Description	Fee ($)	Small Entity Fee ($)
Each claim over 20 (including Reissues)	52	26
Each independent claim over 3 (including Reissues)	220	110
Multiple dependent claims	390	195

Total Claims		Extra Claims		Fee ($)		Fee Paid ($)
40	- 20 or HP =	4	x	26	=	104

HP = highest number of total claims paid for, if greater than 20.

Indep. Claims		Extra Claims		Fee ($)		Fee Paid ($)
3	- 3 or HP =	0	x	110	=	0

HP = highest number of independent claims paid for, if greater than 3.

Multiple Dependent Claims Fee ($)	Multiple Dependent Claims Fee Paid ($)

3. APPLICATION SIZE FEE

If the specification and drawings exceed 100 sheets of paper (excluding electronically filed sequence or computer listings under 37 CFR 1.52(e)), the application size fee due is $270 ($135 for small entity) for each additional 50 sheets or fraction thereof. See 35 U.S.C. 41(a)(1)(G) and 37 CFR 1.16(s).

Total Sheets		Extra Sheets		Number of each additional 50 or fraction thereof		Fee ($)		Fee Paid ($)
	- 100 =		/ 50 =		(round up to a whole number) x		=	

4. OTHER FEE(S)

	Fees Paid ($)
Non-English Specification, $130 fee (no small entity discount)	
Other (e.g., late filing surcharge): Petition Fee ($810)	810

SUBMITTED BY

Signature	David Lewis	Registration No. (Attorney/Agent) 33,101	Telephone (408) 993-1800
Name (Print/Type)	David Lewis		Date Aug 24 2009

PTO/SB/17 (10-08)
Approved for use through 06/30/2010. OMB 0651-0032
U.S. Patent and Trademark Office; U.S. DEPARTMENT OF COMMERCE

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FEE TRANSMITTAL
For FY 2009

☑ Applicant claims small entity status. See 37 CFR 1.27

TOTAL AMOUNT OF PAYMENT ($) 914

Complete if Known	
Application Number	10/527,931
Filing Date	August 26, 2005
First Named Inventor	Adrian I. Bot
Examiner Name	WEHBE, ANNE MARIE SABRINA
Art Unit	1633
Attorney Docket No.	8114-008-WO-US

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☑ Check ☐ Credit Card ☐ Money Order ☐ None ☐ Other (please identify): _____

☑ Deposit Account Deposit Account Number: 503345 Deposit Account Name: _____

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40	- 20 or HP =	4	x	26	=	104

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Indep. Claims		Extra Claims		Fee ($)		Fee Paid ($)
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Multiple Dependent Claims Fee ($)	Fee Paid ($)

3. APPLICATION SIZE FEE

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Total Sheets		Extra Sheets		Number of each additional 50 or fraction thereof		Fee ($)		Fee Paid ($)
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4. OTHER FEE(S)

	Fees Paid ($)
Non-English Specification, $130 fee (no small entity discount)	
Other (e.g., late filing surcharge): Petition Fee ($810)	810

SUBMITTED BY

Signature	David Lewis	Registration No. (Attorney/Agent) 33,101	Telephone (408) 993-1800
Name (Print/Type)	David Lewis		Date Aug 24 2009

I certify that this correspondence was (1) placed in an envelope addressed to

Attention: Office of Petitions
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P.O. Box 1450
Alexandria, Virginia 22313-1450,

and (2) deposited with the United States Post Office with sufficient postage for first class mail on or before the date of

August 24, 2009.

David Lewis

Serial Number:	10/527,931
Filing Date:	August 26, 2005
First Named Inventor:	Adrian I. Bot
Art Unit:	1633
Confirmation Number:	7472
Docket Number:	8114-008-WO-US
Examiner:	WEHBE, ANNE MARIE SABRINA
Title:	Methods and compositions to generate and control the effector profile of t cells by simultaneous loading and activation of selected subsets of antigen presenting cells

Attention: Office of Petitions
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Alexandria, Virginia 22313-1450

Petition to Revive the Application and Explanation of Papers Attached

The Applicants petition that the present application be revived.

Attached are (1) a check for $914 which includes $810 for the petition fee, (2) a response to the Office Action mailed January 28, 2008, and (3) Exhibits I-IV.

This application is currently abandoned due to inadvertently overlooking the deadline for responding to the Office Action of January 28, 2008. Below are an explanation of attached Exhibits I-IV, a summary of the events that led to the deadline for responding to the Office Action being overlooked, and an explanation of the inadvertent delay in filing this petition.

08/28/2009 CCHAU1 00000024 10527931
02 FC:2202 104.00 OP

Exhibit I

Exhibit I is a collection of e-mails and letters evidencing that (1) the Applicants inadvertently overlooked the deadline to file a response to the Office Action and the need to file a petition to revive the application, and (2) that there was an ongoing effort to revive the application from the time that the Applicants became aware of the need to file a petition to revive the application until the filing of this petition. Regarding the letter of September 24, 2008 from Townsend & Townsend & Crew to Multicell Technologies, which is included in Exhibit I, the letter contains attorney client privileged information that is unrelated to current application, and the information that was unrelated to the current application was redacted.

Exhibit II

Exhibit II is an affidavit from W. Gerald Newmin, the President and Chief Executive Officer of the Assignee, which evidences that (1) the failure to respond to the Office Action was inadvertent, (2) that the need to file a petition to revive the application was inadvertently overlooked by the Applicants, and (3) that there was an ongoing effort to revive the application from the time that the Applicants became aware of the need to file a petition to revive the application.

Exhibit III

Exhibit III is an affidavit from the Applicants' Consultant for managing the Applicants' patent portfolio, which evidences that (1) the failure to respond to the Office Action was inadvertent, (2) that the need to file a petition to revive the application was inadvertently overlooked by the Applicants' Consultant, and (3) that there was an ongoing effort to revive the application from the time that the Applicants' Consultant became aware of the need to file a petition to revive the application.

Exhibit IV

Exhibit IV is an affidavit from the Applicants' Representative for Docketing which evidences that there was an ongoing effort to revive the application from the time that the Applicants' Representative realized that the abandonment of the present application was unintentional, until the filing of this petition.

Description of events leading to the unintentional abandonment and unintentional delay in filing this petition

On February 6, 2008 and June 17, 2008 the Applicants received reminders from Catalyst Law Group, informing the Applicants that an Office Action was due for the current application.

However, in early July 2008, due to an accumulation of issues that are not important to this petition, Multicell Technologies Inc. began seeking a new law firm for handling its portfolio of approximately 100 patent applications, and the Applicants lost track of this application and forgot that the Office Action needed to be responded to. As an aside, but for proper context, of the approximately 100 patent applications, 21 were abandoned U.S. or foreign utility applications, 13 were expired P.C.T. applications, 7 were expired U.S. provisional patent applications, 23 were pending or granted U.S. utility patent applications, and 53 were pending or granted foreign patent applications (nearly all of the abandoned or expired applications are parents of pending applications, and consequently the manager of the patent portfolio needs to devote some time to the abandoned and expired patent applications in order to properly manage patent applications that descended from the abandoned and expired applications).

Later in July 2008, Multicell Technologies Inc. selected Townsend and Townsend and Crew as its new representatives, and began the process of transferring its portfolio of approximately 100 patent cases from Catalyst Law Group, APC to Townsend and

Townsend and Crew. Although during this period deadline for reply to the Office Action came due, the Applicants was not focused on this issue, and it was the Applicants' understanding that (1) information, instructions and deadlines for handling pending items related to its patent cases would be transferred from Catalyst Law Group, APC to Townsend and Townsend and Crew and (2) that one of the two law firms would take care of any matters that needed to be taken care of.

In the email from September 2, 2008, the Applicants' Consultant Martin Schroeder states "We have decided to allow 8114-008 to go abandoned ONLY in Canada. I do not recall seeing any rejections from any patent office," which evidences that he is not aware of receiving the rejection in the current application 8114-008US, which is the client's docket number for the current application, which in turn indicates that he is unaware that 8114-008US was abandoned, which (in combination with the affidavits of Martin Schroeder and W. Gerald Newmin) further evidences that the abandonment was unintentional.

Thus, following the transfer and as a result of the transfer of Multicell Technologies Inc.'s patent applications to Townsend and Townsend and Crew (and of the large number of cases) the Applicants were no longer aware of any deadlines for the current application, and did not know the current application had gone abandoned.

Although on September 24, 2008, the Applicants received a letter from Townsend and Townsend and Crew listing proposed strategies for several of Multicell Technologies Inc.'s U.S. and foreign applications, which mentioned docket numbers 8114-007, 8114-008, and 8114-009, and although this letter included the statement, "The U.S. application in this family (USSN 10/527,931) has been abandoned, whereas no examination report has been received in other jurisdictions," the Applicants were relying on Townsend, Townsend and Crew to take appropriate action and for calendaring and tracking

individual applications requiring special attention. The -009 and -007 patent families, mentioned in the letter, contain 11 applications and 10 applications respectively, and additionally, as mentioned above, Multicell's entire patent portfolio contains over 100 applications, which the Applicants were overseeing. The Applicants were not aware of many of the requirements necessary revive this application, such as responding to an Office Action or other requirements.

As evidenced by the statements in the affidavits of Martin Schroeder and W. Gerald Newmin, the Applicants did not recall prior to receiving the September 24, 2008 letter that the current application was abandoned. As evidenced by the statements in the affidavits of Martin Schroeder and W. Gerald Newmin, the Applicants had intended to have the application revived, but were focused on other applications. Further, in the e-mail of June 17, 2009, Martin Schroeder states "Further Adrian's e-mail below and recommendation, let's allow 8114-011 to go abandoned on a worldwide basis and focus on 8114-008," which (in conjunction with the statements in the affidavits from Martin Schroeder and W. Gerald Newmin) further evidences that soon after September 24, 2008, Multicell had forgotten that the application was abandoned and needed to be revived. Additionally the e-mail from June 17, 2009 11:01 A.M. Martin Schroeder states that "It is my understanding that Townsend & Townsend revived 8114-008," which further evidences that Multicell had intended to revive this application and thought it had been revived. Townsend & Townsend and Crew had taken care of. Consequently, since the Applicants had not received further reminders from the prior law firm, taking the necessary steps to revive this application was overlooked, and the fact that it was abandoned was forgotten (as Multicell thought the application had been revived).

On February 4, 2009, Multicell Technologies Inc. hired David Lewis to handle the docketing of Multicell Technologies Inc.'s patent applications. As part of having

information about the Applicants' patent applications entered into docketing software, the statuses of the patent applications were noted and recorded by the Applicants' Representative for docketing David Lewis. It was the assumption of the Applicants' Representative for docketing that all cases labeled as abandoned in docket report prepared by the Applicants' prior firm, Townsend and Townsend and Crew, had been intentionally abandoned.

On June 17, 2009, as mentioned above, Multicell Technologies Inc.'s Consultant Martin Schroeder sent a correspondence to Multicell Technologies Inc.'s representative for docketing patent applications regarding making amendments to the claims of the current application, which was mentioned above.

Also on June 17, 2009, Multicell Technologies Inc.'s representative for docketing had a correspondence sent to inform Multicell Technologies Inc.'s Consultant Martin Schroeder that the current application was abandoned due to a failure to respond to an Office Action, and that a response to the Office Action would be required as part of reviving the application.

Multicell Technologies Inc.'s European patent attorney Richard Clegg, who was assigned to be Multicell's lead attorney for all of its applications worldwide, was preparing claims for the European counterpart to the current application, which the Applicants planned to adapt for use in the current application. Although Richard Clegg's claims were eventually significantly modified based on U.S. practice, Richard Clegg's claims were needed as a basis for the claims in the response to the Office Action necessary for preparing this petition.

Additionally, at that time, Multicell Technologies Inc. was in the process of obtaining a U.S. representative with a background in biotechnology, and did not have anyone for preparing a response to the Office Action in the current applicants, or

adapting the claims being prepared by the European patent attorney. Although David Lewis has a PhD in physics, David Lewis does not ordinarily handle the writing of Biotechnology patent applications or the responses to the Office Actions in Biotechnology patent applications.

Multicell Technologies Inc.'s Consultant Martin Schroeder forwarded e-mail correspondences between Multicell Technologies Inc. and its former attorneys to the Applicants' Representative for docketing, David Lewis.

On June 19, 2009, the Applicants continued efforts to obtain a U.S. biotech attorney or agent, and claims for the European counterpart to the current application were being prepared.

Also on June 19, 2009, David Lewis sent an Employee Agreement and Non Disclosure Agreement to Jennifer Haynes, a registered patent agent with a PhD in microbiology. Although Jennifer Haynes was contracted by David Lewis, Multicell Technologies had not yet agreed to have Jennifer Haynes work on Multicell applications.

June 20, 2009 and June 21, 2009 were a Saturday and a Sunday.

On June 22, 2009, Multicell Technologies Inc.'s Consultant Martin Schroeder forwarded additional e-mail correspondences between Multicell Technologies Inc. and its former attorneys to the Applicants' Representative for docketing, David Lewis. The Applicants continued efforts to obtain a U.S. attorney or agent with a background in biotechnology.

Also on June 22, 2009, claims for the European counterpart to the current application were being prepared.

On June 23, 2009, the Applicants continued efforts to obtain a U.S. attorney or agent with a background in biotechnology. The claims for the European counterpart to

the current application were being prepared by the Applicants' European patent attorney, and the Applicants received and approved of the prepared claims.

On June 24, 2009, Multicell interviewed Jennifer Haynes.

On June 25, 2009 and June 26, 2009, the Applicants continued efforts to obtain a U.S. attorney or agent with a background in biotechnology. The claims for the European counterpart to the current application were being prepared by the Applicants' European patent attorney, and the Applicants received and approved of the prepared claims.

On June 29, 2009, the Applicants' efforts to obtain a U.S. biotech attorney or agent for preparing a response to the Office Action continued. The claims for the European counterpart to the current application were filed in the European application, and the Applicants received a copy of the filed claims.

Thus, the Applicants' Representative David Lewis was waiting for a final version of the claims from June 17, 2009 to June 29, 2009, which were necessary for preparing the response to the Office Action, which is needed for preparing this petition.

Also on June 29, 2009, the Applicants' Representative David Lewis requested approval to begin work on the Petition to Revive and also requested approval for Jennifer Haynes to begin preparing the response to the Office Action in the current application.

Also on June 29, 2009, the Applicants' Consultant gave approval for preparing the Petition to revive the current application. The Applicants' Consultant Martin Schroeder approved of the request. However, there was a miscommunication and the source of the miscommunication is not clear to the Applicants' Representative, but may have been due, in part, to poor a mobile network connection at the Applicants' Consultant's mobile communications device.

On June 30, 2009 – July 2, 2009, the Applicants' Representative David Lewis, continued the review of correspondences between Multicell Technologies Inc. and its

former representatives for information related to the events that contributed to the unintentional abandonment of the current application.

July 3, 2009 and July 4, 2009 were holidays. July 5, 2009 was a Sunday.

On July 6, 2009, the Applicants' Representative began preparing this petition.

On July 7, 2009, the Applicants' Representative continued to work on preparing this petition.

On July 8, 2009, the Applicants' Representative continued to work on preparing this petition.

On July 9, 2009, the Applicants' Representative continued to work on preparing this petition.

On July 15, 2009, the Applicants' Representative David Lewis had a teleconference with the Applicants' Consultant during which the Petition to Revive the current application was discussed, and the incidents leading to the unintentional abandonment of the application were discussed. The Applicants' Representative again requested that approval be given to Jennifer Haynes to begin preparing the response to the Office Action for the current application. The Applicants' Consultant informed the Applicants' Representative David Lewis that approval for Jennifer Haynes to begin preparing the response to the Office Action for the current application had been given in the earlier teleconference on June 29, 2009. The Applicants' Representative David Lewis then realized that he had misunderstood the outcome of the earlier teleconference.

Also on July 15, 2009, the Applicants' Representative had the documents necessary for modifying the European claims and performing other work involved in preparing the response to the Office Action of the current application forwarded to Jennifer Haynes.

On July 16, 2009, Jennifer Haynes began reviewing and preparing documents necessary for filing the response to the Office Action.

July 18, 2009 and July 19, 2009 were Saturday and Sunday.

On July 17, 2009, Applicants' Representative David Lewis and Jennifer Haynes discussed the claims and strategy for responding to the Office Action, and Jennifer Haynes reviewed the claims of the European counterpart of the current application.

On July 20, 2009, Applicants' Representative David Lewis and Jennifer Haynes discussed the Office Action, claims and strategy for responding to the Office Action.

On July 21, 2009, Jennifer Haynes worked on preparing the response to the Office Action.

On July 22, 2009, Jennifer Haynes discussed the claims with the Applicants' Representative David Lewis, and revised the claims.

On July 23, 2009, Jennifer Haynes worked on preparing the response to the Office Action.

On July 24 2009, Jennifer Haynes worked on preparing the response to the Office Action.

July 25, 2009 and July 26, 2009 were Saturday and Sunday.

On July 27 2009, Jennifer Haynes worked on preparing the response to the Office Action.

On July 28 2009, Jennifer Haynes forwarded a draft of the Office Action to the Applicants' Representative David Lewis, for his review.

On July 29 2009, the Applicants' Representative David Lewis forwarded his comments regarding the response to the Office Action to Jennifer Haynes.

On July 30 2009, the Applicants' Representative David Lewis reviewed the revised draft of response to the Office Action and made modifications.

On July 31 2009 – August 10, 2009, Jennifer Haynes worked on preparing the response to the Office.

On August 11, 2009, a draft of the response to the Office Action was forwarded to the Applicants' lead patent attorney, Richard Clegg.

On August 12, 2009, the response to the Office Action was revised.

On August 13, 2009, the response to the Office Action was revised.

On August 14, 2009, a revised draft of the response to the Office Action was forwarded to the Applicants' lead patent attorney, Richard Clegg.

Thus, from June 29, 2009 to August 14, 2009 the response to the Office Action, which was necessary for filing this petition, was being prepared.

August 15, 2009 and August 16, 2009 were Saturday and Sunday.

On August 17, 2009, the Applicants' Representative David Lewis continued work on preparing this petition.

On August 18, 2009, the Applicants' Representative David Lewis continued work on preparing this petition.

On August 19, 2009, the Applicants' Representative David Lewis continued work on preparing this petition.

On August 20, 2009, the Applicants' Representative David Lewis continued work on preparing this petition.

On August 21, 2009, the Applicants' Representative David Lewis continued work on preparing this petition.

August 22, 2009 and August 23, 2009 were Saturday and Sunday.

On August 24, 2009, the Applicants' Representative David Lewis worked on and filed this petition.

Summary of why the abandonment and the entire delay in filing this petition was unintentional

Thus, to summarize, from June 17, 2009 from June 29, 2009, the Applicant's Representative was waiting to receive the final claims from the European counterpart of the present application, in addition to other activities that were occurring related to preparing this petition. Although a registered U.S. practitioner had been approved by Martin Schroeder on June 29, 2009, due to a miscommunication, David Lewis was not aware of the approval and had not assigned writing the response to the Office Action until July 15, 2009. Despite the miscommunication, other activities related to reviving the current application did occur between June 29, 2009 and July 15, 2009. From July 15, 2009 to August 14, 2009 the response to the Office Action, which was necessary for filing this petition, was being prepared, in addition to other activities that were occurring related to preparing this petition. From August 14, 2009 to August 20, 2009, this petition was being prepared. Thus from June 17, 2009 to August 20, 2009 the Applicants were diligently working on activities related to preparing this petition, and any delay in preparing the petition during that period was unintentional.

Whereas regarding prior to June 17, 2009, the abandonment was inadvertent due to Multicell being distracted with its other applications and a change in the law firms representing them, and Multicell was not aware of the abandonment until around September 24, 2009 when informed by Townsend & Townsend and Crew. However, Multicell was distracted with other matters, and assumed that Townsend & Townsend and Crew would take care of the necessary steps required to revive the application. Consequently Multicell inadvertently overlooked the reviving of the application, and Multicell did not discover that the application was still abandoned and had never been revived until June 17, 2009.

Conclusion

Thus, the entire delay in filing the petition to revive was inadvertent and the Applicants respectfully submit that it would be proper to grant the present petition, and the Applicants respectfully requests that the present application be revived.

Respectfully Submitted,

David Lewis

Aug 24 2009
Date

Registration Number 33,101
1250 Aviation Avenue, Suite 200B
San Jose, California 95110

EXHIBIT I

E-mails and Letters

Kari Moyer-Henry, Ph.D.
kmoyerhenry@catalystlaw.com
(858) 200-0591

February 6, 2008

Via E-mail: jnewmin@aol.com

Jerry Newmin
MultiCell Technologies, Inc.
701 George Washington Highway
Lincoln, RI 02865

Re: U.S. Patent Application Serial No.: 10/527,931
"METHODS AND COMPOSITIONS TO GENERATE AND CONTROL THE EFFECTOR PROFILE OF T CELLS BY SIMULTANEOUSLY LOADING AND ACTIVATION OF SELECTED SUBSETS OF ANTIGEN PRESENTING CELLS"
Filed August 26, 2005
Our File No. 8114-008-WO-US

Dear Mr. Newmin:

Enclosed please find a copy of an Office Action we received for the above referenced application. The deadline to file a response is **April 28, 2008**. Please review the examiner's comments and let us have your instructions for filing our response.

Please contact our office if you have any questions.

Very truly yours,
CATALYST LAW GROUP, APC

Kari Moyer-Henry, Ph.D.
Intellectual Property Clerk

kmh/DMK
Enclosure
Cc: M. Schroeder (via e-mail)

Henderson R. Stansbury; Patent Firm of David Lewis

From: Martin Schroeder [martin_schroeder@emmesgroup.com]
Sent: Monday, June 22, 2009 1:54 PM
To: Henderson Stansbury; David Lewis
Subject: FW: Need instructions for following matters
Follow Up Flag: Follow up
Flag Status: Blue
Categories: MULTICELL TECHNOLOGIES

-----Original Message-----
From: David Kohn [mailto:DKohn@catalystlaw.com]
Sent: Tuesday, June 17, 2008 1:37 PM
To: martin_schroeder@emmesgroup.com; Jerry Newmin
Cc: Lucille Gomes; Tom Jurgensen; Kari Moyer-Henry; Jazmine Lumanlan
Subject: Need instructions for following matters

Jerry/Martin,

Per our reminder letters, we have yet to receive instructions for the following matters:

8114-007-WO-US (DNA case) – final deadline to respond is June 21, 2008 (3 extensions of time);
8114-008-US (DNA case) – deadline to respond is June 28, 2008 (2 extensions of time; final deadline for response due July 28, 2008);
8114-005-CON (IgG case) – shortened statutory period for response due June 30, 2008 (no extensions; final deadline for response due September 30, 2008)

Martin, I believe you guys are working on the response for the 8114-005-CON application, but I wanted to bring your attention to the first two. Please let me know, as these will require advance payments for the government costs associated with the response.

Regards,
David

David M. Kohn | Catalyst Law Group APC
9710 Scranton Rd., Suite 170 | San Diego, CA 92121
t. 858.200.0586 | f. 858.450.9834 | c. 858.735.0843
dkohn@catalystlaw.com | www.catalystlaw.com

Henderson R. Stansbury; Patent Firm of David Lewis

From: Martin Schroeder [martin_schroeder@emmesgroup.com]
Sent: Monday, June 22, 2009 1:59 PM
To: Henderson Stansbury; David Lewis
Subject: FW: Multicell 8114 Patent Families
Follow Up Flag: Follow up
Flag Status: Blue
Categories: MULTICELL TECHNOLOGIES
Attachments: RomeroEtAl_10550722.pdf; 8114-009 USPTO Rejection.pdf; CD3 Antibody Therapy to Treat Type 1 Diabetes.pdf; hOKT3 MAb Type 1 Diabetes Columbia Kevan Herold.pdf; MultiCell Immunotherapeutics 06-236.pdf; MultiCell Immunotherapeutics Gantt Chart 06-236.doc1.pdf; Bot JOI 2006 IgTAA paper.pdf; Phillips Smith IgPeptide Delivery IRI 2005.pdf

-----Original Message-----
From: Martin Schroeder [mailto:martin_schroeder@emmesgroup.com]
Sent: Tuesday, September 02, 2008 6:58 PM
To: cgao@townsend.com; Ken Weber
Cc: Jerry Newmin; jnewmin@multicelltech.com; Lucille Gomes
Subject: Multicell 8114 Patent Families

Ken, Chaun,

I have again reviewed the 8114-007, -008, -009, and -011 Multicell patent families, and have the following comments/questions/recommendations.

(1) 8114-007 Patent Family

This patent family originates from PCT application PCT/US03/07995. There are two pending corresponding US patent applications: US serial numbers 11/585,588 and 10/507,942. The US patent office rejected 10/507,942, and the European patent office rejected the EP equivalent to PCT/US03/07995. I have no information about the status of US patent application 11/585,588. Surprisingly, the Chinese patent granted the Chinese equivalent of PCT/US03/07995 with amended claims. This patent family relates to the use of non-coding RNA in conjunction with an antigen alone, or with an antigen contained in the CDR of an antibody to improve the immune response.

The two cited US patent applications seem to have exactly the same claims, so I am not sure why there are two apparently unique US patent applications covering the same invention. Can you clarify?

(2) 8114-008 Patent Family

This patent family originates from PCT/US03/30188. There is one pending corresponding US patent application serial number 10/527,931. This patent family relates to ways to generate an enhanced T-cell response in patients to an antigen wherein at least one epitope portion of the antigen in contained in the CDR of an antibody and administering the antibody-antigen construct in conjunction with dsRNA. The proposed claims appear to be limited to specific groups of peptide epitopes, and a molecular weight range of the dsRNA (10-50KD), and describes the up/down regulation of certain Interleukins and Interferons. The proposed claims also discuss the induction of an "effective memory response" to the administered peptide epitope and an enhanced Th1 and Th2 response.

We have decided to allow 8114-008 to go abandoned ONLY in Canada. I do not recall seeing any rejections from any patent office. I would appreciate it if you can determine the status of the US and EP equivalents of PCT/US03/30188, and let me know if there have been any rejection notices.

(3) 8114-009 Patent Family

This patent family originates from PCT/US04/09261. There is one pending US patent application serial number 10/550,722. This patent family relates to the use of dsRNA or ssRNA alone to cause apoptosis in proliferating cells. The claimed novelty seems to lie in the use of dsRNA (pA:pU) or ssRNA (pA), is less than 50KD in molecular weight, the proliferating cells are cancer cells, and that administration pA:pU or pA of less than 10KD causes enhanced alpha-TNF production. In case you are not aware, administering anything to a human that causes increased production of alpha-TNF can potentially be life threatening, so what is truly novel in my opinion is the finding that pA:pU or pA that is greater than 20KD still seems to cause apoptosis in cancer cells, enhanced T-1 immune response and IL-12 production, but DOES NOT cause the increased production of alpha-TNF. [See Locksley RM, Killeen N, Lenardo MJ, "The TNF and TNF receptor superfamilies: integrating mammalian biology", Cell 104 (4): 487–501 (2001).] This finding concerning not provoking an alpha-TNF response with pA:pU or pA that is greater than 20KD could be very important since many siRNA drugs currently in development I believe are all less than 10KD.

The USPTO rejected 10/550,722 (attached) for a number of reasons including the attached Romero et. al. reference. We need a game plan to figure out how to amend the claims if possible so that they will be allowed by the USPTO.

With respect to the 8114-007, -008, -009 patent families discussed above, I refer to David Kohn's emails of June 20, 2008 wherein David recommends: "...submit new claims or file continuation applications off of the -007 and -008 applications (given the breadth of their disclosures relative to the -009 application) and still receive the benefit of the Sept 2002 priority date". I do not know if David's recommendation is the appropriate course of action that Multicell should follow. Please evaluate David's recommendation, and let me know your recommendation if we should proceed according to David's recommendations with respect to the 8114-007, -008, and -009 patent families, or take a different approach. Feel free to contact David directly should you wish to discuss the matter with him.

(4) 8114-011 Patent Family

This patent family originates from PCT/US06/032512. There is no corresponding US patent application. I am not sure why. The international search report accompanying WO 2007/022477 A3 lists numerous references which are presented as prior art with the effect of negating the novelty of the proposed claims. The international search report was compiled on July 11, 2007. This PCT relates to a means to enhance antigen loading on the antigen presenting cell (APC) with the goal of enhancing the antigen's presentation on the MHC class 1 molecule to improve the desired T-cell response. This PCT specifically focuses on "tolerization" of a person to self-antigens such as would be the case in multiple sclerosis, type-1 diabetes and other such autoimmune diseases. The specific idea is to craft the self-antigen peptide sequence into the CDR of a therapeutic antibody, then administer the grafted therpeutic antibody construct to a patient suffering from the autoimmune disease. Multicell has data in mouse models for multiple sclerosis and type-1 diabetes showing that the disease phenotype is 100% ameliorated following administration of the corresponding self-antigen therapeutic antibody construct. Hence, the idea of "tolerization" to self-antigens. I have attached two references that show alternative therapeutic antibody approaches to treating type-1 diabetes, a couple of Multicell's own journal articles discussing their technology, and Multicell's grant applications with respect to the use of the claimed invention for the treatment of juvenile type-1 diabetes.

I look forward to hearing your detailed recommendations on how we should proceed in due course.

Thanks again for your help.

Martin

****

Martin Schroeder
EVP & Managing Director
Direct: (415) 495-7111
Mobile: (510) 816-3515
FAX: (415) 495-3777

TOWNSEND
and
TOWNSEND
and
CREW
LLP

San Francisco

Two Embarcadero Center
Eighth Floor
San Francisco, California 94111-3834
Tel 415.576.0200
Fax 415.576.0300

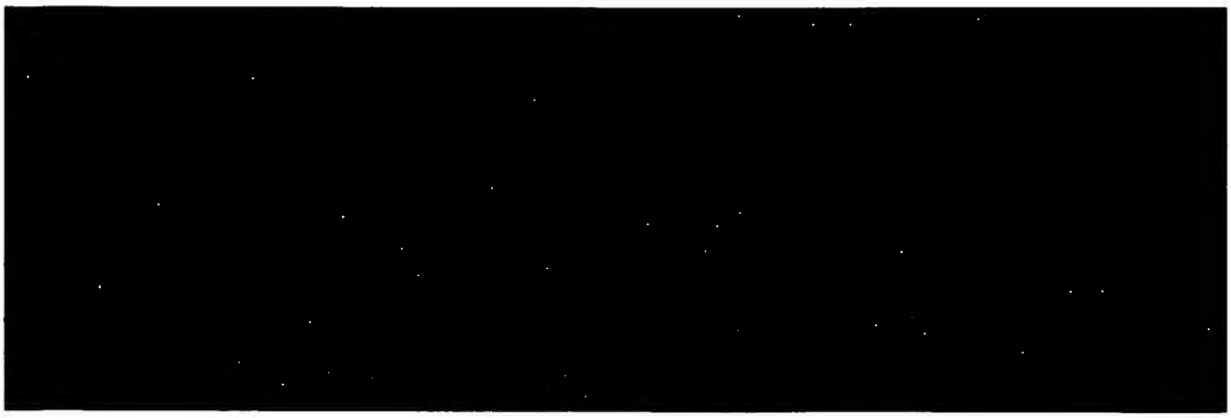
CONFIDENTIAL ATTORNEY-CLIENT PRIVILEGED

September 24, 2008

Jerry Newmin
MultiCell Technologies, Inc.
701 George Washington Highway
Lincoln, RI 02865

RE: Patent Prosecution Strategy
027607-000600 family (Multicell 8114-7 series)
027607-000700 family (Multicell 8114-8 series)
027607-000800 family (Multicell 8114-9 series)

Dear Mr. Newmin:



027607-000600 (8114-7): [redacted]

[redacted]

027607-000700 (8114-8): [redacted]

[redacted]

A. If you intend to further confirm the patentability of these proposed claims, we can perform a patentability analysis at the minimum cost of about $2,500 to $5,000, depending on the state of the art. Please let us know if you wish to proceed with the patentability analysis.

B. If you approve of the proposed new claims as outlined above, we will proceed to take necessary steps in the corresponding U.S. and foreign applications to replace the currently pending claims with these new claims. The U.S. application in this family (USSN 10/527,931) has been abandoned, whereas no examination report has been received in other jurisdictions. Assuming that the abandonment of the U.S. application was unintentional, we propose to petition for its revival as soon as possible so that the new claims can be submitted. The estimated cost for reviving this application is $6,000. Likewise, we will submit the new claims upon receiving the first examination report in the foreign applications. Please let us know if you approve of filing of the proposed claims.

027607-000800 (8114-9): [redacted]

[redacted]

Henderson R. Stansbury; Patent Firm of David Lewis

From: David Lewis [davidlewisnmn@yahoo.com]
Sent: Monday, July 20, 2009 3:33 PM
To: jennifer.a.haynes@comcast.net
Cc: 'Henderson R. Stansbury; David Lewis Patent Firm'
Subject: RE: 74-89 claims
Attachments: 74-89 some proposed claims.doc

Dear Jennifer,

See my claim 1A. I am not sure if it makes sense (e.g., whether it is supported by the spec and whether it is a more detailed version of the method referenced in Richard Clegg's claims). If it makes sense, I think we should amend claim 1, as outlined in claim 1A. We should add claim 1B, and then add the rest of Richard's claims. However, it appears to me that Richard's claims need to be modified for US practice - we should discuss that over the phone.

The idea of claim 1A is just to give the Examiner something that builds on the previous claims. Claim 1B is a broader version of claim 1A. this claim I am hoping will link claim 1 to the composition/antigen claims that the rest of the claims will be amended to be (either by amending them or by inserting new ones).

In any event Martin is more interested in the composition of matter than the method, because he believes that he will get the best protection that way (which logically makes some sense, but if you have any thoughts about that, let me know). I nonetheless think it would be a good idea to have a couple of method claims mirroring the composition claims to round out their patent protection a bit (that should only cost them an extra $220). I am not sure how to claim the antigen, because it seems that part of the invention may be the use of the antigen in combination with other compounds. In the mechanical world, I might claim a "kit," which is not always well received by the Examiners, but is sometimes the only way to claim certain inventions and in theory should work. Although I can think of analogies to kits that might work, perhaps there is a better (e.g, well established) way in the biotech world of claiming a multiple compounds that are used together (?we may want to also have claims for the mixture of the two compounds?). We should discuss this.

Please feel free to contact me regarding anything you would like to discuss.

Best regards,

David Lewis

Phone: 408-993-1800
Fax: 408-993-1800

1250 Aviation Avenue, Suite 200B
San Jose, California 95110

http://www.davidlewispatentagent.com

To send a fax, just call 408-993-1800 at anytime. If you get my recorded message, just press start or send on your fax machine, and my answering service is supposed to automatically detect that there is an incoming fax and receive it. If for some reason this does not work, please let me know. Thanks!

Henderson R. Stansbury; Patent Firm of David Lewis

From: David Lewis [davidlewisnmn@yahoo.com]
Sent: Tuesday, July 21, 2009 10:40 AM
To: jennifer.a.haynes@comcast.net
Subject: Richard's claims

Dear Jennifer,

If you find any other claims that we should ask Richard about, please let me know. Note that the Europeans have "use" claims, which are expressly mentioned in the EPC rules, which are not quite our method of use (because typically the word "method" would not even appear in a "use" claims), and which I do not fully understand.

Thanks!

Please feel free to contact me regarding anything you would like to discuss.

Best regards,

David Lewis

Phone: 408-993-1800
Fax: 408-993-1800

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Henderson R. Stansbury; Patent Firm of David Lewis

From: Jennifer Haynes [jennifer.a.haynes@comcast.net]

Sent: Tuesday, July 21, 2009 11:30 AM

To: davidlewisnmn@yahoo.com

Subject: Re: Richard's claims

Hi David, yes, use claims are odd. I'm staying away from them. I do have some questions about the word "synthetic" before dsRNA. It looked like there was a lot of discussion with the EU associate about whether they should take that word out. I think your idea about the "kit" claims is a great idea. I hope he likes it too. I should have a first draft set of claims done by this afternoon...should I email it to you to review? Because the US does not allow those mixed agent/use claims that they did in the EU claim set, I ended up with quite a bit fewer claims. It also seemed like all of the claims in the PCT and EU included both the agent and the RNA, so I did not prepare an agent only claim. I will recheck this, though. Lastly, I have a couple of method claims (that are dependent on the kit and/or composition claims) and I am working on a dependent claim for these that discusses in vivo and ex vivo use. The original PCT claims had something like this. OK, let me know. Thanks, Jen

Henderson R. Stansbury; Patent Firm of David Lewis

From: David Lewis [davidlewisnmn@yahoo.com]
Sent: Tuesday, July 21, 2009 12:02 PM
To: 'Jennifer Haynes'
Subject: RE: Richard's claims

Dear Jennifer,
See my comments below.

Thanks!

Please feel free to contact me regarding anything you would like to discuss.

Best regards,

David Lewis

Phone: 408-993-1800
Fax: 408-993-1800

1250 Aviation Avenue, Suite 200B
San Jose, California 95110

http://www.davidlewispatentagent.com

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From: Jennifer Haynes [mailto:jennifer.a.haynes@comcast.net]
Sent: Tuesday, July 21, 2009 11:30 AM
To: davidlewisnmn@yahoo.com
Subject: Re: Richard's claims

Hi David, yes, use claims are odd. I'm staying away from them. I do have some questions about the word "synthetic" before dsRNA. It looked like there was a lot of discussion with the EU associate about whether they should take that word out. -

>>I assume there is support in the specification for the word "synthetic." Do we need it for patentability and what does it add? The word synthetic seems something like a product-by-process limitation.

I think your idea about the "kit" claims is a great idea. I hope he likes it too.

>> Probably, they don't use the word "kit" in the specification. I am wondering if, depending on how the specification is written, we should call it something else like a "medical product for administering a treatment" and then have a dependent claim that says that the agent and the

dsRNA are in separate containers/viles, if supported by the specification.

I should have a first draft set of claims done by this afternoon...should I email it to you to review? -

>>That works

Because the US does not allow those mixed agent/use claims that they did in the EU claim set, I ended up with quite a bit fewer claims. It also seemed like all of the claims in the PCT and EU included both the agent and the RNA, so I did not prepare an agent only claim.

>>In mechanical/electrical US applications, from the point of view of 35 USC 112/132, I have yet to have troubles with adding a claim that is broader than any of the original claims. Have you had problems with doing that in Biotech cases?

>>It would seem that it might be worth a try. What do you think?

I will recheck this, though. Lastly, I have a couple of method claims (that are dependent on the kit and/or composition claims) and I am working on a dependent claim for these that discusses in vivo and ex vivo use.

>> in other words, you mean whether the agent and ds RNA are mixed ex vivo or administered together (and therefore not mixed until they are in vivo).

The original PCT claims had something like this.

>>If we can do this while more or less sticking to the language of the original application, that would be great!

OK, let me know. Thanks, Jen

----- Original Message -----
From: David Lewis
To: jennifer.a.haynes@comcast.net
Sent: Tuesday, July 21, 2009 10:40 AM
Subject: Richard's claims

Dear Jennifer,
If you find any other claims that we should ask Richard about, please let me know. Note that the Europeans have "use" claims, which are expressly mentioned in the EPC rules, which are not quite our method of use (because typically the word "method" would not even appear in a "use" claims), and which I do not fully understand.

Thanks!

Please feel free to contact me regarding anything you would like to discuss.

Best regards,

David Lewis

Phone: 408-993-1800
Fax: 408-993-1800

1250 Aviation Avenue, Suite 200B
San Jose, California 95110

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To send a fax, just call 408-993-1800 at anytime. If you get my recorded message, just press start or send on your fax machine, and my answering service is supposed to automatically detect that there is an incoming fax and receive it. If for some reason this does not work, please let me know. Thanks!

Henderson R. Stansbury; Patent Firm of David Lewis

From: Jennifer Haynes [jennifer.a.haynes@comcast.net]

Sent: Tuesday, July 21, 2009 3:38 PM

To: davidlewisnmn@yahoo.com

Subject: Re: Richard's claims

Attachments: NEW US CLAIMS.doc

Hi David, thanks for the comments, here is what I have so far...let's discuss this claims set and a possible broader claim when you have a chance. I looked at the sequence listing rejection and once I have the final listing that was submitted in the PCT case I can amend the figure legends to include the SEQ ID NOs (that way we won't have to amend the figures themselves - which simplifies things). But I won't know until I see it. I have also prepared a draft of the response which is awaiting our final claim draft. Call anytime, Jen

Henderson R. Stansbury; Patent Firm of David Lewis

From: David Lewis [davidlewisnmn@yahoo.com]
Sent: Tuesday, July 21, 2009 4:17 PM
To: 'Jennifer Haynes'
Subject: RE: Richard's claims
Attachments: NEW US CLAIMS v2.doc

Dear Jennifer,

I will call you soon.

Please feel free to contact me regarding anything you would like to discuss.

Best regards,

David Lewis

Phone: 408-993-1800
Fax: 408-993-1800

1250 Aviation Avenue, Suite 200B
San Jose, California 95110

http://www.davidlewispatentagent.com

To send a fax, just call 408-993-1800 at anytime. If you get my recorded message, just press start or send on your fax machine, and my answering service is supposed to automatically detect that there is an incoming fax and receive it. If for some reason this does not work, please let me know. Thanks!

Henderson R. Stansbury; Patent Firm of David Lewis

From: Jennifer Haynes [jennifer.a.haynes@comcast.net]

Sent: Wednesday, July 22, 2009 11:37 AM

To: davidlewisnmn@yahoo.com

Cc: jennifer.a.haynes@comcast.net

Subject: version 2 claims

Attachments: NEW US CLAIMSversion2.doc

Hi David, here is the next version. Let me know if I forgot anything. I will be out for a bit at noon, but should be available by about 2:30 if you want to talk about it. I added back your claim 1 with a couple of changes..see what you think....thanks, Jen

Henderson R. Stansbury; Patent Firm of David Lewis

From: David Lewis [davidlewisnmn@yahoo.com]
Sent: Wednesday, July 22, 2009 2:05 PM
To: 'Jennifer Haynes'
Subject: RE: version 2 claims
Attachments: 74-89 NEW US CLAIMS version4.doc

Dear Jennifer,

Most of the changes are fairly minor, see if you like amended claim 1. I removed the last two claims.

Please feel free to contact me regarding anything you would like to discuss.

Best regards,

David Lewis

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Fax: 408-993-1800

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San Jose, California 95110

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Henderson R. Stansbury; Patent Firm of David Lewis

From: Jennifer Haynes [jennifer.a.haynes@comcast.net]

Sent: Wednesday, July 22, 2009 4:29 PM

To: davidlewisnmn@yahoo.com; Henderson R. Stansbury; David Lewis Patent Firm

Subject: sequence listing

Hi David and Henderson,

I was just reviewing the sequence listing rejection in the OA and it is possible that we can fix it with some amendments to the specification (if the sequence listing that was submitted in the parent application has all of the sequences in it). Henderson, can you help me get a copy of the parent sequence listing? Then I can review it to make sure all of the sequences are there. Thanks, I really appreciate it! Jen

Henderson R. Stansbury; Patent Firm of David Lewis

From: Henderson R. Stansbury; David Lewis Patent Firm [henderson@davidlewispatentagent.com]
Sent: Wednesday, July 22, 2009 4:43 PM
To: jennifer.a.haynes@comcast.net
Cc: davidlewisnmn@yahoo.com
Subject: FW: sequence listing

No problem. David mentioned it earlier, but I didn't get a chance to check all the possible places I might find it. I'll keep looking.

From: Jennifer Haynes [mailto:jennifer.a.haynes@comcast.net]
Sent: Wednesday, July 22, 2009 4:29 PM
To: davidlewisnmn@yahoo.com; Henderson R. Stansbury; David Lewis Patent Firm
Subject: sequence listing

Hi David and Henderson,
I was just reviewing the sequence listing rejection in the OA and it is possible that we can fix it with some amendments to the specification (if the sequence listing that was submitted in the parent application has all of the sequences in it). Henderson, can you help me get a copy of the parent sequence listing? Then I can review it to make sure all of the sequences are there. Thanks, I really appreciate it! Jen

Henderson R. Stansbury; Patent Firm of David Lewis

From: David Lewis [davidlewisnmn@yahoo.com]
Sent: Wednesday, July 22, 2009 7:12 PM
To: 'Jennifer Haynes'
Subject: RE: Claims
Attachments: 74-89 NEW US CLAIMS version6.doc

Dear Jennifer,

I had another thought. Usually, I file my applications with a method of use and a method of making, which seems to be in line with the reasons that Martin wanted the "use" claims - although he clearly never asked for a method of making. in any even I added one method of making claim - its the last claim. It does not say much more than we are making the product of claim 41. See if you like it.

Please feel free to contact me regarding anything you would like to discuss.

Best regards,

David Lewis

Phone: 408-993-1800
Fax: 408-993-1800

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San Jose, California 95110

http://www.davidlewispatentagent.com

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Henderson R. Stansbury; Patent Firm of David Lewis

From: Jennifer Haynes [jennifer.a.haynes@comcast.net]

Sent: Tuesday, July 28, 2009 1:27 PM

To: davidlewisnmn@yahoo.com

Cc: Henderson R. Stansbury; David Lewis Patent Firm

Subject: Re: EPO Sequence Listing

Hi David, 3 things:

1. SEQLIST - I think you are right. I think we need to get a text copy of the EP listing from the EP associate or the client if that is possible. Then we could change the application number to fit the US and submit that. Do you think that would be possible? It would save the client a lot of money rather than having us do it from scratch.
2. OA response - I have a draft of the response which I can send to you today or tomorrow. I'd love your thoughts on strategy, etc. But, it is not done - it is only a first draft. Would you rather I wait until it is more finalized?
3. References - I was able to get a copy of the two patents that are cited, but could not get the ref. Cella et al. (1999) J. Exp. Med. Vol 189 (5), 821-829. I would like to do a quick review. Do you guys have a way of getting these types of references? Also, I would love to see what the references D1 and D2 are for the EP case. Would you and Henderson be able to help me with either or both of these? Let me know.

Thanks for all of your help! Jen

Henderson R. Stansbury; Patent Firm of David Lewis

From: Henderson R. Stansbury; David Lewis Patent Firm [henderson@davidlewispatentagent.com]
Sent: Tuesday, July 28, 2009 2:18 PM
To: jennifer.a.haynes@comcast.net
Cc: davidlewisnmn@yahoo.com
Subject: Re: EPO Sequence Listing - References

Hi Jen,

Could you send me the identifying information for references D1 and D2, or a copy of the page that lists them? I can't quite find anything listed under those exact labels.

Please let me know if we can be of further assistance to you in any way.

Take care,

HENDERSON R. STANSBURY
ADMIN ASSISTANT TO DAVID LEWIS Ph.D

DAVID LEWIS PATENT FIRM
PHONE: (408) 993-1800 | FAX: (408) 993-1800
1250 Aviation Avenue, Suite 200B
San Jose, California 95110
http://www.davidlewispatentagent.com

To send a fax, just call 408-993-1800 at anytime. If you get my recorded message, just press start or send on your fax machine, and my answering service is supposed to automatically detect that there is an incoming fax and receive it.

If for some reason this does not work, please let me know. Thanks!

Henderson R. Stansbury; Patent Firm of David Lewis

From: David Lewis [davidlewisnmn@yahoo.com]
Sent: Tuesday, July 28, 2009 8:44 PM
To: 'Jennifer Haynes'
Cc: 'Henderson R. Stansbury; David Lewis Patent Firm'
Subject: resp to OA
Attachments: 74-89 resp to oa v2 7-21-09.doc; x-xx (BM-TEMPLATE) Response to Office Action.doc

Dear Jennifer,
Overall I like it, but see my comments. The passwords will be sent in a subsequent e-mail

I use the following naming convention, "docket# [applic,resp to OA, etc.] version_number" Feel free to add whatever you want either before or after the version number. Always increment the version number after making any change so as not to get earlier and later version confused with one another.

I should have sent you my template for responses to Office Actions. As you have already written the response, don't worry too much about following it, but I would like to add the certificate of mailing to the front page. If there is anything else you like in it feel free to use it. Shouldn't we amend the specification to include the sequence listing? Or, is that not the way it is done?

The template that I am sending you is somewhat corrupted with actual data from another patent application. I will have Henderson send you a better version when he gets a chance to correct that. Most of the fields are supposed to get filled in automatically from our database.

Please feel free to contact me regarding anything you would like to discuss.

Best regards,

David Lewis

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Fax: 408-993-1800

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San Jose, California 95110

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Henderson R. Stansbury; Patent Firm of David Lewis

From: Henderson R. Stansbury; David Lewis Patent Firm [henderson@davidlewispatentagent.com]
Sent: Wednesday, July 29, 2009 10:19 AM
To: jennifer.a.haynes@comcast.net
Cc: davidlewisnmn@yahoo.com
Subject: Re: EPO Sequence Listing

Dear Jen,

We plan on asking Richard for the text version. David is in a conference call and he will get back to you soon.

Please let me know if we can be of further assistance to you in any way.

Take care,

HENDERSON R. STANSBURY
ADMIN ASSISTANT TO DAVID LEWIS Ph.D

DAVID LEWIS PATENT FIRM
PHONE: (408) 993-1800 | FAX: (408) 993-1800
1250 Aviation Avenue, Suite 200B
San Jose, California 95110
http://www.davidlewispatentagent.com

To send a fax, just call 408-993-1800 at anytime. If you get my recorded message, just press start or send on your fax machine, and my answering service is supposed to automatically detect that there is an incoming fax and receive it.

If for some reason this does not work, please let me know. Thanks!

Henderson R. Stansbury; Patent Firm of David Lewis

From: Henderson R. Stansbury; David Lewis Patent Firm [henderson@davidlewispatentagent.com]
Sent: Wednesday, July 29, 2009 11:53 AM
To: jennifer.a.haynes@comcast.net
Cc: davidlewisnmn@yahoo.com
Subject: 8114-008EP References D1 and D2
Attachments: D2-03797931_EN8TOH9U8453472.pdf; D1-US5969109A.pdf

<<...>> <<...>>

Hi Jen,

Attached are the references you were looking for.

Please let me know if we can be of further assistance to you in any way.

Take care,

HENDERSON R. STANSBURY
ADMIN ASSISTANT TO DAVID LEWIS Ph.D

DAVID LEWIS PATENT FIRM
PHONE: (408) 993-1800 | FAX: (408) 993-1800
1250 Aviation Avenue, Suite 200B
San Jose, California 95110
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If for some reason this does not work, please let me know. Thanks!

Henderson R. Stansbury; Patent Firm of David Lewis

From: David Lewis [davidlewisnmn@yahoo.com]
Sent: Wednesday, July 29, 2009 12:22 PM
To: 'Jennifer Haynes'
Subject: RE: resp to OA - call me when it is convenient

Dear Jennifer,

I think my comment about claim 77 was erroneous. I must have misread what the Examiner wrote.

Please feel free to contact me regarding anything you would like to discuss.

Best regards,

David Lewis

Phone: 408-993-1800
Fax: 408-993-1800

1250 Aviation Avenue, Suite 200B
San Jose, California 95110

http://www.davidlewispatentagent.com

To send a fax, just call 408-993-1800 at anytime. If you get my recorded message, just press start or send on your fax machine, and my answering service is supposed to automatically detect that there is an incoming fax and receive it. If for some reason this does not work, please let me know. Thanks!

Henderson R. Stansbury; Patent Firm of David Lewis

From: David Lewis [davidlewisnmn@yahoo.com]
Sent: Wednesday, July 29, 2009 1:24 PM
To: 'Jennifer Haynes'
Cc: 'Henderson R. Stansbury; David Lewis Patent Firm'
Subject: RE: newest draft
Attachments: 74-89 resp to oa v4 7-21-09 (2).doc

Dear Jennifer,

I made a couple of more changes, see if you like them. Also, one of the headings in the response to the Office Action is "Notice to Comply," but I did not see a notice comply that was being replied to.

Please feel free to contact me regarding anything you would like to discuss.

Best regards,

David Lewis

Phone: 408-993-1800
Fax: 408-993-1800

1250 Aviation Avenue, Suite 200B
San Jose, California 95110

http://www.davidlewispatentagent.com

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Henderson R. Stansbury; Patent Firm of David Lewis

From: Henderson R. Stansbury; David Lewis Patent Firm [henderson@davidlewispatentagent.com]
Sent: Friday, July 31, 2009 9:42 AM
To: jennifer.a.haynes@comcast.net
Cc: davidlewisnmn@yahoo.com
Subject: Sequence Listing in text formats, and Cella reference update
Attachments: Sequence listing.doc; Sequence listing.txt

<<...>> <<...>>

Hi Jen,

Attached are copies of the Sequence Listing in .doc and .txt formats (not that there is anything that needs to be done with them that we are aware of, just keeping you in the loop).

I left a message for the Examiner regarding the Cella reference, and if I don't hear back from her in a couple of hours I'll try calling her again to request a copy of the reference.

Have a good weekend.

Please let me know if we can be of further assistance to you in any way.

Take care,

HENDERSON R. STANSBURY
ADMIN ASSISTANT TO DAVID LEWIS Ph.D

DAVID LEWIS PATENT FIRM
PHONE: (408) 993-1800 | FAX: (408) 993-1800
1250 Aviation Avenue, Suite 200B
San Jose, California 95110
http://www.davidlewispatentagent.com

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If for some reason this does not work, please let me know. Thanks!

Henderson R. Stansbury; Patent Firm of David Lewis

From: David Lewis [davidlewisnmn@yahoo.com]
Sent: Friday, July 31, 2009 11:22 AM
To: 'Jennifer Haynes'
Subject: RE: newest draft
Attachments: article 821.pdf

Dear Jennifer,

Here is the article. I will wait for you to review it, before sending the draft of the response to the Office Action to Martin Schroeder and Richard Clegg.

Please feel free to contact me regarding anything you would like to discuss.

Best regards,

David Lewis

Phone: 408-993-1800
Fax: 408-993-1800

1250 Aviation Avenue, Suite 200B
San Jose, California 95110

http://www.davidlewispatentagent.com

To send a fax, just call 408-993-1800 at anytime. If you get my recorded message, just press start or send on your fax machine, and my answering service is supposed to automatically detect that there is an incoming fax and receive it. If for some reason this does not work, please let me know. Thanks!

Henderson R. Stansbury; Patent Firm of David Lewis

From: David Lewis [davidlewisnmn@yahoo.com]

Sent: Monday, August 03, 2009 5:13 PM

To: adrian1bot@aol.com; jennifer.a.haynes@davidlewispatentagent.com

Cc: martin_schroeder@emmesgroup.com; 'Jerry Newmin'; 'Lucille Gomes'; 'Henderson R. Stansbury; David Lewis Patent Firm'

Subject: MultiCell's ref. #8114-008US, Methods and compositions to generate and control the effector profile of T Cells by simultaneous loading and activation of selected subsets of antigen presenting cells

Attachments: Cella et al.pdf

Dear Adrian,

I would like to introduce you to Jennifer, who is working with me on a response to an Office Action in the above application. Jennifer's email address is jennifer.a.haynes@davidlewispatentagent.com.

Jennifer would like to get you input regarding the above reference, which was not cited in the corresponding European patent application, but is being used in a rejection in the US application. If you would like a copy of application and/or claims, please let me know.

Please feel free to contact me regarding anything you would like to discuss.

Best regards,

David Lewis

Phone: 408-993-1800
Fax: 408-993-1800

1250 Aviation Avenue, Suite 200B
San Jose, California 95110

http://www.davidlewispatentagent.com

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Henderson R. Stansbury; Patent Firm of David Lewis

From: adrian1bot@aol.com

Sent: Tuesday, August 04, 2009 11:10 AM

To: davidlewisnmn@yahoo.com; jennifer.a.haynes@davidlewispatentagent.com

Cc: martin_schroeder@emmesgroup.com; jnewmin@aol.com; lgomes@multicelltech.com; henderson@davidlewispatentagent.com

Subject: Re: MultiCell's ref. #8114-008US, Methods and compositions to generate and control the effector profile of T Cells by simultaneous loading and activation of selected subsets of antigen presenting cells

Dear David,

Thank you ! I am looking forward to working with Jennifer on this one. I appreciate a lot the latest version of claims.

Regards,

Adrian

Henderson R. Stansbury; Patent Firm of David Lewis

From: Martin Schroeder [martin_schroeder@emmesgroup.com]

Sent: Tuesday, August 04, 2009 1:00 PM

To: davidlewisnmn@yahoo.com; adrian1bot@aol.com; jennifer.a.haynes@davidlewispatentagent.com

Cc: 'Jerry Newmin'; 'Lucille Gomes'; 'Henderson R. Stansbury; David Lewis Patent Firm'

Subject: RE: MultiCell's ref. #8114-008US, Methods and compositions to generate and control the effector profile of T Cells by simultaneous loading and activation of selected subsets of antigen presenting cells

Everyone,

I want to reinforce that Richard Clegg at Mewburn is the lead patent attorney for all Multicell patent cases. To that end, 8114-008US needs to comport to what Richard and Fran have already done on 8114-008EP. Please coordinate your efforts and include Richard in all of your discussions.

Thanks,

Martin

****

Martin Schroeder
EVP & Managing Director
Emmes Group
Tel: (415) 495-7111
Mobile: (510) 816-3515
FAX: (415) 495-3777

Henderson R. Stansbury; Patent Firm of David Lewis

From: David Lewis [davidlewisnmn@yahoo.com]
Sent: Tuesday, August 04, 2009 2:05 PM
To: martin_schroeder@emmesgroup.com
Cc: jennifer.a.haynes@davidlewispatentagent.com; 'Richard Clegg'; adrian1bot@aol.com
Subject: RE: 8114-008 (74-89)

Dear Martin,

Since the next time that Adrian is available at 8:00 am is August 12, and since we need to minimize the delay in filing the petition to revive, would it be O.K., if Jennifer spoke to both Adrian and Richard, but separately, regarding Cella et al. (e.g., she would first speak to Adrian and then to Richard to get his input and to make sure that he is totally in agreement), so that we can finalize the response to the Office Action and petition earlier than August 12?

Thanks!

Please feel free to contact me regarding anything you would like to discuss.

Best regards,

David Lewis

Phone: 408-993-1800
Fax: 408-993-1800

1250 Aviation Avenue, Suite 200B
San Jose, California 95110

http://www.davidlewispatentagent.com

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Henderson R. Stansbury; Patent Firm of David Lewis

From: David Lewis [davidlewisnmn@yahoo.com]
Sent: Tuesday, August 04, 2009 2:07 PM
To: 'Jennifer Haynes'; jennifer.a.haynes@davidlewispatentagent.com
Subject: RE: clarification: teleconferences and discussion with Adrian

Dear Jennifer,

I don't need to participate in any discussion that just involves you and Adrian. I would like to be in discussions that involve Richard, at least for now.

Please feel free to contact me regarding anything you would like to discuss.

Best regards,

David Lewis

Phone: 408-993-1800
Fax: 408-993-1800

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San Jose, California 95110

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Henderson R. Stansbury; Patent Firm of David Lewis

From: Martin Schroeder [martin_schroeder@emmesgroup.com]

Sent: Tuesday, August 04, 2009 2:08 PM

To: davidlewisnmn@yahoo.com

Cc: jennifer.a.haynes@davidlewispatentagent.com; 'Richard Clegg'; adrian1bot@aol.com

Subject: RE: 8114-008 (74-89)

David,

Yes. It is OK. Just make sure that if you need to file claims to revive 8114-008, that those claims are consistent with the claims file with the EPO for 8114-008EP.

Martin

****

Martin Schroeder
EVP & Managing Director
Emmes Group
Tel: (415) 495-7111
Mobile: (510) 816-3515
FAX: (415) 495-3777

Henderson R. Stansbury; Patent Firm of David Lewis

From: David Lewis [davidlewisnmn@yahoo.com]
Sent: Tuesday, August 04, 2009 2:23 PM
To: martin_schroeder@emmesgroup.com
Cc: jennifer.a.haynes@davidlewispatentagent.com; 'Richard Clegg'; adrian1bot@aol.com
Subject: RE: 8114-008 (74-89)

O.K. - David

Please feel free to contact me regarding anything you would like to discuss.

Best regards,

David Lewis

Phone: 408-993-1800
Fax: 408-993-1800

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San Jose, California 95110

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EXHIBIT II

Affidavit of W. Gerald Newmin

I certify that this correspondence was (1) placed in an envelope addressed to

Attention: Office of Petitions
Mail Stop Petition
Commissioner for Patents
P.O. Box 1450
Alexandria, Virginia 22313-1450,

and (2) deposited with the United States Post Office with sufficient postage for first class mail on or before the date of

July Aug 24, 2009.

David Lewis

Serial Number:	10/527,931
Confirmation Number:	7472
Filing Date:	August 26, 2005
Examiner:	WEHBE, ANNE MARIE SABRINA
Art Unit:	1633
First Named Inventor:	Adrian I. Bot
Docket Number:	8114-008-WO-US
Title:	Methods and compositions to generate and control the effector profile of t cells by simultaneous loading and activation of selected subsets of antigen presenting cells

Attention: Office of Petitions
Mail Stop Petition
Commissioner for Patents
P.O. Box 1450
Alexandria, Virginia 22313-1450

Affidavit of W. Gerald Newmin

I am W. Gerald Newmin, and I reside at

[3224-C Sunset Key Circle

[Punta Gorda, FL] 33955

I am the President and Chief Executive Officer of Multicell Technologies Inc., which is located at

68 Cumberland Street, Suite 301
Woonsocket, RI 02895.

As President of Multicell Technologies Inc., I authorized the filing of the current application, which was filed on August 26, 2005.

I have essentially no training in prosecuting applications before the United States Patent and Trademark Office.

On February 6, 2008, I received a letter from Catalyst Law Group, APC informing me that an Office Action had been mailed by the U.S. Patent and Trademark for the current application.

On June 17, 2008, I received an e-mail from David Kohn at Catalyst Law Group, APC requesting instructions for the pending Office Action, and informing me that the deadline for responding to Office Action was July 28, 2008.

In early July 2008, due to an accumulation of issues that are not important to this petition, Multicell Technologies Inc. began seeking a new law firm for handling our portfolio of approximately 100 patent applications.

Later in July 2008, Multicell Technologies Inc. selected Townsend and Townsend and Crew as its new representatives, and began the process of transferring our portfolio of approximately 100 patent cases from Catalyst Law Group, APC to Townsend and Townsend and Crew. Although during this period deadline for reply to the Office Action came due, I was not focused on this issue, and it was my understanding that information, instructions and deadlines for handling pending items related to our patent cases would be transferred from Catalyst Law Group, APC to Townsend and Townsend and Crew and that one of the two law firms would take care of any matters that needed to be taken care of.

Any failure to meet deadlines and requirements for this application during the period of time when Multicell Technologies Inc.'s patent applications were being transferred between firms was unintentional. Following the transfer of Multicell Technologies Inc.'s patent applications to Townsend and Townsend and Crew I was no longer aware of any deadlines for the current application, and I did not know the current application had gone abandoned.

On September 24, 2008, I received a letter from Townsend and Townsend and Crew listing proposed strategies for several of Multicell Technologies Inc.'s U.S. and foreign applications, which mentioned docket numbers 8114-007, 8114-008, and 8114-009. Although this letter included the statement, "The U.S. application in this family (USSN 10/527,931) has been abandoned, whereas no examination report has been received in other jurisdictions," I was relying on Townsend, Townsend and Crew to take appropriate action and for calendaring and tracking individual applications requiring special attention. The -009 and -007 patent families, mentioned in the letter, contain 11 applications and 10 applications respectively, and additionally Multicell's entire patent portfolio contains over 100 applications, which I was overseeing. I was not aware of the requirement to respond to an Office Action or what other requirements would need to be met to revive the application, and I did not know prior to receiving this letter that the current application was abandoned. I had intended to have the application revived, but this application was not the primary focus of my attention. Consequently, since I did not get any further reminders, seeing to it that this application was revived was overlooked, and the fact that it was abandoned was forgotten.

On February 4, 2009, Multicell Technologies Inc. hired David Lewis to handle the docketing of Multicell Technologies Inc.'s patent applications.

On June 16, 2009, Multicell Technologies Inc.'s Consultant Martin Schroeder copied me on a correspondence with Multicell Technologies Inc.'s representative for docketing patent applications regarding an amendment to the claims of the current application.

On June 17, 2009, Multicell Technologies Inc.'s representative for docketing copied me on a correspondence informing Multicell Technologies Inc.'s Consultant Martin Schroeder that the current application was abandoned due to a failure to respond to an Office Action, and that a response to the Office Action would be required as part of reviving the application.

Multicell Technologies Inc.'s European patent attorney was preparing claims for the European counterpart to the current application, which we planned to adapt for use in the current application. However, Multicell Technologies Inc. was in the process of obtaining a U.S. representative with a background in biotech at that time, and did not have anyone capable of preparing a response to the Office Action, and adapting the claims being prepared by the European patent attorney.

Multicell Technologies Inc.'s Consultant Martin Schroeder forwarded e-mail correspondences between Multicell Technologies Inc. and its former attorneys to the Applicant's Representative for docketing, David Lewis.

On June 19, 2009, we continued efforts to obtain a U.S. biotech attorney or agent, and claims for the European counterpart to the current application were being prepared.

June 20, 2009 and June 21, 2009 were a Saturday and a Sunday.

On June 22, 2009, Multicell Technologies Inc.'s Consultant [Martin Schroeder] forwarded e-mail correspondences between Multicell Technologies Inc. and its former attorneys to the Applicant's Representative for docketing, David Lewis. We continued efforts to obtain a U.S. biotech attorney or agent, and claims for the European counterpart to the current application were being prepared.

On June 23, 2009 – June 26, 2009, we continued efforts to obtain a U.S. biotech attorney or agent. The claims for the European counterpart to the current application were being prepared by the Applicant's European patent attorney, and I received and approved of the prepared claims.

On June 29, 2009, we continued efforts to obtain a U.S. biotech attorney or agent for preparing a response to the Office Action continued.

The claims for the European counterpart to the current application were filed in the European application, and I received a copy of the filed claims.

Any delay in filing the petition to revive the current application resulting from me was completely unintentional.

I hereby declare that all statements made herein of my own knowledge are true and that all statements made on information and belief are believed to be true; and further that these statements were made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under 18 U.S.C. 1001 and that such willful false statements may jeopardize the validity of the application or any patent issued thereon.

Respectfully Submitted,

8/4/09
Date

W. Gerald Newmin
President & Chief Executive Officer
Multicell Technologies, Inc.

EXHIBIT III

Affidavit of Applicants' Consultant Martin Schroeder

I certify that this correspondence was (1) placed in an envelope addressed to

Attention: Office of Petitions
Mail Stop Petition
Commissioner for Patents
P.O. Box 1450
Alexandria, Virginia 22313-1450,

and (2) deposited with the United States Post Office with sufficient postage for first class mail on or before the date of

July Aug 24, 2009, 2009.

David Lewis

Serial Number:	10/527,931
Confirmation Number:	7472
Filing Date:	August 26, 2005
Examiner:	WEHBE, ANNE MARIE SABRINA
Art Unit:	1633
First Named Inventor:	Adrian I. Bot
Docket Number:	8114-008-WO-US
Title:	Methods and compositions to generate and control the effector profile of t cells by simultaneous loading and activation of selected subsets of antigen presenting cells

Attention: Office of Petitions
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Commissioner for Patents
P.O. Box 1450
Alexandria, Virginia 22313-1450

Affidavit of Martin Schroeder

I am Martin Schroeder, and I reside at:

92 Natoma Street, Suite 200
San Francisco, CA 94105

I am a consultant to Multicell Technologies Inc., which is located at

68 Cumberland Street, Suite 301
Woonsocket, RI 02895.

As a consultant Multicell Technologies Inc., I provide assistance in making decisions relevant to the management of Multicell Technologies Inc.'s patent portfolio.

I have essentially no training in prosecuting applications before the United States Patent and Trademark Office.

On February 6, 2008, I received a letter from Catalyst Law Group, APC informing me that an Office Action had been mailed by the U.S. Patent and Trademark for the current application.

On June 17, 2008, I received an e-mail from David Kohn at Catalyst Law Group, APC requesting instructions for the pending Office Action, and informing me that the deadline for responding to Office Action was July 28, 2008.

In early July 2008, Multicell Technologies Inc. began seeking new representatives for handling our portfolio of approximately 100 patent applications.

Later in July 2008, Multicell Technologies Inc. selected Townsend and Townsend and Crew as its new representatives, and began the process of transferring our portfolio of approximately 100 patent cases from Catalyst Law Group, APC to Townsend and Townsend and Crew. Although during this period, the response to the Office action was due, it was my understanding that information, instructions and deadlines for handling pending items related to our patent cases would be transferred from Catalyst Law Group, APC to Townsend and Townsend and Crew. Since I did not receive any reminders and since this application was not the focus of my attention, responding to the Office Action was inadvertently overlooked.

Any failure to meet deadlines and requirements for this application during the period of time when Multicell Technologies Inc.'s patent applications were being transferred between firms was unintentional. Following the transfer of Multicell Technologies Inc.'s patent applications to Townsend and Townsend and Crew I was no

longer aware of any deadlines for the current application, and I did not know the current application was abandoned.

On September 2, 2008, I sent an e-mail to Townsend and Townsend and Crew related to patent prosecution strategies for several applications, including the present application. I informed Townsend and Townsend and Crew that I did not recall seeing any rejection from the U.S. Patent and Trademark Office in the current application and that only the Canadian variant of the current application should be allowed to go abandoned. I was not aware of the requirement to respond to an Office Action, and I did not know the current application was abandoned.

On September 24, 2008, I received a letter from Townsend and Townsend and Crew listing proposed strategies for several of Multicell Technologies Inc.'s U.S. and foreign applications, which mentioned docket numbers 8114-007, 8114-008, and 8114-009. Although this letter included the statement, "The U.S. application in this family (USSN 10/527,931) has been abandoned, whereas no examination report has been received in other jurisdictions," I was relying on Townsend, Townsend and Crew to take appropriate action and for calendaring and tracking individual applications requiring special attention. The -009 and -007 patent families, mentioned in the letter, contain 11 applications and 10 applications respectively, and additionally Multicell's entire patent portfolio contains over 100 applications, which I was overseeing. Since I did not receive any further reminders and since this application was not the focus of my attention, reviving the application was inadvertently overlooked. Any failure on my part to provide instructions necessary for taking required actions for the current application was unintentional. The fact that the application was abandoned was forgotten.

On February 4, 2009, Multicell Technologies Inc. hired David Lewis to handle the docketing of Multicell Technologies Inc.'s patent applications.

On June 16, 2009, I corresponded with Multicell Technologies Inc.'s representative for docketing patent applications regarding an amendment to the claims of the current application.

On June 17, 2009, Multicell Technologies Inc.'s representative for docketing informed me that the current application was abandoned due to a failure to respond to an Office Action, and that a response to the Office Action would be required as part of reviving the application.

Multicell Technologies Inc.'s European patent attorney was preparing claims for the European counterpart to the current application, which we planned to adapt for use in the current application. However, Multicell Technologies Inc. was in the process of obtaining a U.S. representative with a background in biotech at that time, and did not have anyone capable of preparing a response to the Office Action, and adapting the claims being prepared by the European patent attorney.

I forwarded e-mail correspondences between Multicell Technologies Inc. and its former attorneys to the Applicant's Representative, David Lewis, who handles docketing.

On June 19, 2009, we continued efforts to obtain a U.S. biotech attorney or agent, and claims for the European counterpart to the current application were being prepared.

On June 22, 2009, I forwarded e-mail correspondences between Multicell Technologies Inc. and its former attorneys to the Applicant's Representative for docketing, David Lewis. We continued efforts to obtain a U.S. biotech attorney or agent, and claims for the European counterpart to the current application were being prepared.

On June 23, 2009, we continued efforts to obtain a U.S. biotech attorney or agent. The claims for the European counterpart to the current application were being prepared by the Applicant's European patent attorney.

On June 24, 2009, I interviewed Jennifer Haynes as part of ongoing efforts to obtain a U.S. representative for preparing responses to the Office Actions in U.S. patent applications, including this application.

June 25 – June 26, 2009, we continued efforts to obtain a U.S. biotech attorney or agent. The claims for the European counterpart to the current application were being prepared by the Applicant's European patent attorney, and I received and approved of the prepared claims.

On June 29, 2009, we continued efforts to obtain a U.S. biotech attorney or agent for preparing a response to the Office Action continued.

The claims for the European counterpart to the current application were filed in the European application, and I received a copy of the filed claims.

The Applicant's Representative, David Lewis, requested approval to begin work on the Petition to Revive and also requested approval for Jennifer Haynes to begin preparing the response to the Office Action in the current application.

I gave approval to begin work on the Petition to Revive the application and the response to the Office Action.

On June 30, 2009 – July 2, 2009, we continued efforts to obtain a U.S. biotech attorney or agent.

On July 15, 2009, I had a teleconference the Applicant's Representative, David Lewis, during which the Petition to Revive the current application was discussed. The

Applicant's Representative again requested that approval be given to Jennifer Haynes to begin preparing the response to the Office Action for the current application. I informed the Applicant's Representative, David Lewis that approval for Jennifer Haynes to begin preparing the response to the Office Action for the current application had been given in an earlier teleconference.

Any delay in filing the petition to revive the current application resulting from me was completely unintentional.

I hereby declare that all statements made herein of my own knowledge are true and that all statements made on information and belief are believed to be true; and further that these statements were made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under 18 U.S.C. 1001 and that such willful false statements may jeopardize the validity of the application or any patent issued thereon.

Respectfully Submitted,

7/29/2009
Date

Martin Schroeder

EXHIBIT IV

Affidavit of Applicants' Representative
David Lewis

I certify that this correspondence was (1) placed in an envelope addressed to

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P.O. Box 1450
Alexandria, Virginia 22313-1450,

and (2) deposited with the United States Post Office with sufficient postage for first class mail on or before the date of

August 24, 2009.

David Lewis

Serial Number:	10/527,931
Confirmation Number:	7472
Filing Date:	August 26, 2005
Examiner:	WEHBE, ANNE MARIE SABRINA
Art Unit:	1633
First Named Inventor:	Adrian I. Bot
Docket Number:	8114-008-WO-US
Title:	Methods and compositions to generate and control the effector profile of t cells by simultaneous loading and activation of selected subsets of antigen presenting cells

Attention: Office of Petitions
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Commissioner for Patents
P.O. Box 1450
Alexandria, Virginia 22313-1450

Affidavit of David Lewis

I am a registered patent agent with a PhD in physics and I have not filed responses to rejections or written patent applications for inventors in the field of biotechnology.

On February 4, 2009, I was hired by Multicell Technologies Inc. to handle the Docketing of the Applicants' patent applications. I was forwarded a docket report prepared by Townsend and Townsend and Crew containing information about the applicants' patent applications. As part of entering information about the Applicants' patent applications into our docketing software, the statuses of the patent applications were noted and recorded. It was my understanding that all cases labeled as abandoned in

docket report prepared by the Applicants' prior firm, Townsend and Townsend and Crew, had been intentionally abandoned.

On June 16, 2009, I received an e-mail from the Applicants' Consultant for managing the Applicants' patent portfolio, Martin Schroeder. The Applicants' Consultant's e-mail stating that the Applicants were planning to amend the claims of the current application.

On June 17, 2009, as part of my standard procedure, I reviewed basic information about the current application, including the status of the current application, before making comments or a recommendation on proceeding. While reviewing the case information, I noticed that the current application was abandoned.

Also on June 17, 2009, I discussed the abandonment of the application with the Applicants' Consultant, and the Applicants' Consultant was unaware that the application had gone abandoned. I explained to the Applicants' Consultant that the application went abandoned due to a failure to respond to an Office Action, and that a response to the Office Action would be required as part of reviving the application.

Also on June 17, 2009, the Applicants' Consultant informed me via e-mail that the Applicants' European patent attorney was preparing claims for the European counterpart to the current application, which the Applicants planned to adapt for use in the current application.

Also on June 17, 2009, the Applicants' Consultant forwarded me e-mail correspondences between Multicell Technologies Inc. and its former attorneys, and I reviewed the correspondences.

June 20, 2009 and June 21, 2009 were a Saturday and a Sunday.

On June 22, 2009, I received an e-mail from the Applicants' Consultant which contained copies of e-mail correspondences between Multicell Technologies Inc. and its former attorneys, and I reviewed the correspondences.

On June 23, 2009, the Applicants' efforts to obtain a U.S. representative for preparing a response to the Office Action continued. The claims for the European counterpart to the current application were being prepared by the Applicants' European patent attorney.

On June 24, 2009, I participated in an interview, in which the Applicants' Consultant interviewed Jennifer Haynes, a registered patent agent.

June 25 – June 26, 2009, the Applicants' efforts to obtain a U.S. representative for preparing a response to the Office Action continued. The claims for the European counterpart to the current application were being prepared by the Applicants' European patent attorney, and the claims were sent to the Applicants and the Applicants' Consultant for review and approval.

June 27, 2009 and June 28, 2009 were a Saturday and a Sunday.

On June 29, 2009, the Applicants' Representative, David Lewis, requested approval to begin work on the Petition to Revive and also requested approval for Jennifer Haynes to begin preparing the response to the Office Action in the current application.

Also on June 29, 2009, the Applicants' Consultant gave approval for preparing the Petition to revive the current application, but it was not clear to the Applicants' Representative, David Lewis that approval had been given for Jennifer Haynes to begin work on the response to the Office Action. The miscommunication regarding approval for Jennifer Haynes to begin working on the response to the Office Action may have been

due, in part, to poor a mobile network connection at the Applicants' Consultant's mobile communications device.

On June 30, 2009 – July 2, 2009, I continued the review of correspondences between Multicell Technologies Inc. and its former representatives for information related to the events that contributed to the unintentional abandonment of the current application. The Applicants' efforts to obtain a U.S. representative for preparing a response to the Office Action continued.

July 3, 2009 and July 4, 2009 were holidays. July 5, 2009 was a Sunday.

On July 6, 2009, I began preparing this petition.

On July 7, 2009, I continued to work on preparing this petition.

On July 8, 2009, I continued to work on preparing this petition.

On July 9, 2009, I continued to work on preparing this petition.

On July 15, 2009, the Applicants' Representative David Lewis had a teleconference with the Applicants' Consultant during which the Petition to Revive the current application was discussed. The Applicants' Representative again requested that approval be given to Jennifer Haynes to begin preparing the response to the Office Action for the current application. The Applicants' Consultant informed the Applicants' Representative David Lewis that approval had been given in an earlier teleconference for Jennifer Haynes to begin preparing the response to the Office Action for the current application, and the Applicants' Representative David Lewis then realized that he had misunderstood the outcome of the earlier teleconference.

Also on July 15, 2009, The Applicants' Representative David Lewis had the documents necessary for modifying the European claims and performing other work

involved in preparing the response to the Office Action of the current application forwarded to Jennifer Haynes.

On July 16, 2009, Jennifer Haynes began reviewing and preparing documents necessary for filing the response to the Office Action.

July 18, 2009 and July 19, 2009 were Saturday and Sunday.

On July 17, 2009, I discussed the claims and strategy for responding to the Office Action with Jennifer Haynes.

On July 20, 2009, I discussed the claims and strategy for responding to the Office Action with Jennifer Haynes.

On July 22, 2009, I discussed the claims with Jennifer Haynes.

On July 23, 2009, Jennifer Haynes worked on preparing the response to the Office Action.

On July 24 2009, Jennifer Haynes worked on preparing the response to the Office Action.

July 25, 2009 and July 26, 2009 were Saturday and Sunday.

On July 27, 2009, Jennifer Haynes worked on preparing the response to the Office Action.

On July 28 2009, I received a draft of the Office Action from Jennifer Haynes, and I began reviewing the draft.

On July 29 2009, I forwarded my comments regarding the response to the Office Action to Jennifer Haynes.

On July 30 2009, I reviewed the revised draft of response to the Office Action and made modifications.

On July 31 2009, I worked on revising the response to the Office Action.

August 1, 2009 and August 2, 2009 were Saturday and Sunday.

On August 11, 2009, I forwarded a draft of the response to the Office Action to the Applicants' lead patent attorney, Richard Clegg.

On August 12, 2009, I received the revised draft the response to the Office Action from Jennifer Haynes.

On August 13, 2009, I revised the draft of the response to the Office Action.

On August 14, 2009, I forwarded a draft of the response to the Office Action to the Applicants' lead patent attorney, Richard Clegg.

August 15, 2009 and August 16, 2009 were Saturday and Sunday.

On August 17, 2009, I continued work on preparing this petition.

On August 18, 2009, I continued work on preparing this petition.

On August 19, 2009, I continued work on and filed this petition. On August 21, 2009, the Applicants' Representative David Lewis continued work on preparing this petition.

August 22, 2009 and August 23, 2009 were Saturday and Sunday.

On August 24, 2009, the Applicants' Representative David Lewis worked on and filed this petition.

Any delay in filing the petition to revive the current application resulting from me was completely unintentional.

I hereby declare that all statements made herein of my own knowledge are true and that all statements made on information and belief are believed to be true; and further that these statements were made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under 18 U.S.C. 1001 and that such willful false statements may jeopardize the validity of the application or any patent issued thereon.

Respectfully Submitted,

David Lewis

Aug 24, 2009
Date

Registration Number 33,101
1250 Aviation Avenue, Suite 200B
San Jose, California 95110

PATENT

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

I certify that this correspondence was (1) placed in an envelope addressed to

Attention: Office of Petitions
Mail Stop Petition
Commissioner for Patents
P.O. Box 1450
Alexandria, Virginia 22313-1450,

and (2) deposited with the United States Post Office with sufficient postage for first class mail on or before the date of

August 24, 2009.

David Lewis

Confirmation No. 7472
Examiner: Wehbe, Anne Marie Sabrina
Art Unit: 1633
In re application of: Bot, Adrian, et al.
Application No.: 10/527,931
Filed: August 26, 2005
For: METHODS AND COMPOSITIONS TO GENERATE AND CONTROL THE EFFECTOR PROFILE OF T CELLS BY SIMULTANEOUS LOADING AND ACTIVATION OF SELECTED SUBSETS OF ANTIGEN PRESENTING CELLS

AMENDMENT

Attention: Office of Petitions
Mail Stop Petition
Commissioner for Patents
P.O. Box 1450
Alexandria, Virginia 22313-1450

Sir:

In response to the Office Action mailed January 28, 2008, please enter the following amendments and remarks:

Amendments to the Claims are reflected in the listing of claims which begins on page 2 of this paper.

Amendments to the Specification begin on page 7 of the paper.

Amendments to the Sequence Listing begin on page 12 of the paper.

Remarks/Arguments begin on page 13 of this paper.

Amendments to the Claims:

Please replace the prior set of claims with the claims below, which are redlined to show the changes to the claims.

Listing of Claims:

1. (CURRENTLY AMENDED) A method of generating an enhanced T cell response, with the product of claim 41, in a patient to ~~an~~ the antigen, the method comprising:

administering to the patient an immunoglobulin or portion thereof wherein said immunoglobulin or portion thereof has at least one peptide epitope of said antigen attached to said immunoglobulin or portion thereof; and administering said immunoglobulin or portion thereof in conjunction with ~~a~~ the ~~dsRNA~~ double stranded RNA ~~segment~~ to said patient in an amount sufficient to generate a Tc1 response in the patient to the antigen;

wherein the agent is the IgG having the at least one T cell epitope of the antigen covalently attached within the CDR region of the IgG without modification of the Fc portion.

Cancel Claims 2-36

37. (NEW) A composition comprising: an agent including at least an IgG having at least one T cell epitope of an antigen covalently attached within the CDR region of the IgG without modification of the Fc portion; and

double-stranded RNA.

38. (NEW) The composition of claim 37, wherein the double-stranded RNA is pA:pU.

39. (NEW) The composition of claim 37, wherein the double-stranded RNA is pI:pC.

40. (NEW) The composition according to claim 37 wherein the agent and double-stranded RNA are provided in an amount sufficient to generate a Tc1 response in a patient.

41. (NEW) A product for administering a treatment for virus immunization, viral infection or tumor, comprising: an agent including at least an IgG having at least one T cell epitope of an antigen covalently attached within the CDR region of the IgG without modification of the Fc portion; and

double-stranded RNA; wherein the agent and the double-stranded RNA are provided in an amount sufficient to generate a Tc1 response in a patient.

42. (NEW) The product of claim 41, wherein the double-stranded RNA is pA:pU.

43. (NEW) The product of claim 41, wherein the double-stranded RNA is pI:pC.

44. (NEW) The product of claim 41, wherein the double-stranded RNA is pA:pU and is provided in an amount sufficient to induce MHC class I-restricted Tc1 cells thereby producing IFN-γ.

45. (NEW) The product of claim 41, wherein the double-stranded RNA has a molecular weight of between 10 - 50 Kd.

46. (NEW) The product of claim 41, wherein the double-stranded RNA are between 100 - 2000 base pairs in length.

47. (NEW) The product of claim 41, wherein the immunoglobulin backbone of the IgG is derived from human IgG, or is a humanized IgG.

48. (NEW) The product of claim 41, wherein the patient is human.

49. (NEW) The product of claim 41, wherein the antigen is a virus.

50. (NEW) The product of claim 49, wherein the virus is influenza virus.

51. (NEW) The product of claim 41, wherein the T cell epitope is selected from: influenza virus M1 or M2; hepatitis C virus NS3; hepatitis B virus core antigen; human papilloma virus HPV 18-E7, HPV 16 - E7, HPV 18 E6, HPV 16 E6; HIV-1: reverse transcriptase; HIV-1: gag; herpes simplex antigens; and respiratory syncytial virus antigens.

52. (NEW) The product of claim 41, wherein the T-cell epitope is a tumor associated T cell epitope.

53. (NEW) The product of claim 41, wherein the T cell epitope is selected from: melanoma-gp100; MART-1; TRP-2; carcinoembryonic antigen precursor; Her-2; prostate

tumor antigens; carcinoembryonic antigen precursor XP064845/NCB1; prostate tumor antigens; MUC 1; and mucin 1.

54. (NEW) The product of claim 41, wherein the agent and double-stranded RNA are admixed together.

55. (NEW) The product of claim 41, wherein the agent and double-stranded RNA are provided separately to be administered separately.

56. (NEW) The composition of claim 37, wherein the double-stranded RNA is pA:pU and is provided in an amount sufficient to induce MHC class I-restricted Tc1 cells thereby producing IFN-γ.

57. (NEW) The composition of claim 37, wherein the double-stranded RNA has a molecular weight of between 10 - 50 Kd.

58. (NEW) The composition of claim 37, wherein the double-stranded RNA are between 100 - 2000 base pairs in length.

59. (NEW) The composition of claim 37, wherein the immunoglobulin backbone of the IgG is derived from human IgG, or is a humanized IgG.

60. (NEW) The composition of claim 37, wherein the patient is human.

61. (NEW) The composition of claim 37, wherein the antigen is a virus.

62. (NEW) The composition of claim 61, wherein the virus is influenza virus.

63. (NEW) The composition of claim 37, wherein the T cell epitope is selected from: influenza virus M1 or M2; hepatitis C virus NS3; hepatitis B virus core antigen; human papilloma virus HPV 18-E7, HPV 16 - E7, HPV 18 E6, HPV 16 E6; HIV-1: reverse transcriptase; HIV-1: gag; herpes simplex antigens; and respiratory syncytial virus antigens.

64. (NEW) The composition of claim 37, wherein the T-cell epitope is a tumor associated T cell epitope.

65. (NEW) The composition of claim 37, wherein the T cell epitope is selected from : melanoma-gp100; MART-1; TRP-2; carcinoembryonic antigen precursor; Her-2; prostate tumor antigens; carcinoembryonic antigen precursor XP064845/NCB1; prostate tumor antigens; MUC 1; and mucin 1.

66. (NEW) A viral vaccine comprising a composition according to claim 37.

67. (NEW) A viral vaccine comprising a product according to claim 41.

68. (NEW) A method of virus immunization comprising:

administering the product of claim 41 to a patient in need of virus immunization.

69. (NEW) A method of treatment of viral infection comprising:

administering the product of claim 41 to a patient in an amount sufficient to treat the viral infection.

70. (NEW) A method for treatment of tumor comprising:

administering the product of claim 41 to a patient in need of tumor treatment.

71. (NEW) A method of generating an enhanced T cell response in a patient to an antigen, comprising:

administering the composition of claim 37 to a patient in an amount sufficient to generate a Tc1 response in the patient to an antigen.

72. (NEW) A method of generating an enhanced T cell response in a patient using the product of claim 41, comprising:

administering the agent to a patient in conjunction with double-stranded RNA, in an amount sufficient to generate a Tc1 response in the patient to an antigen.

73. (NEW) The method of claim 72, wherein the agent and the double-stranded RNA are administered together.

74. (NEW) The method of claim 72, wherein the agent and the double-stranded RNA are administered separately.

75. (NEW) The method of claim 72, wherein the agent and double-stranded RNA are administered *in vivo*.

76. (NEW) A method of producing the product of claim 41, comprising:

producing the agent with at least the IgG having at least one T cell epitope of the antigen covalently attached within the CDR region of the IgG without modification of the Fc portion; and

producing the double-stranded RNA; wherein the agent and the double-stranded RNA are provided in an amount sufficient to generate a Tc1 response in a patient.

AMENDMENTS TO THE SPECIFICATION

Please insert the following at line 11:

INCORPORATION BY REFERENCE OF THE SEQUENCE LISTINGS ON COMPACT DISC

This application hereby incorporates by reference two identical files, each entitled "Sequence-Listing.txt," each of the two files is stored on a different one of two identical compact discs, both files were created on August 21, 2009, and both files have the size of 86,924 bytes.

Please replace the paragraph starting on page 18 line 20 and ending at line 21 of the Brief Description of the Drawings, with the following paragraph:

--Fig. 1D shows the sequence of the constant region of the heavy chain as well as schematic depiction of a prospective construct. The construct contains the Human IgG1 backbone Leader and Flanking sequence (Seq. I.D. No. 48), the glycine polylinker (Seq. I.D. No. 49), the Hinge region of human recombinant Ig (Seq. I.D. No. 3), the human IgG1 backbone CH2 region of human recombinant Ig (Seq. I.D. No. 2), and the human IgG1 backbone CH3 region of human recombinant Ig (Seq. I.D. No. 1). The combination sequence containing the Hinge, CH2 and CH3 regions is provided in Seq. I.D. No. 7; --

Please replace the paragraph starting on page 18 line 22 and ending at line 26 of the Brief Description of the Drawings, with the following paragraph:

--Fig. 1E - 1M show the sequences of various antigens and epitopes discussed in the present application and which can be inserted into an immunoglobulin [sequences can be accessed on the internet at ncbi.nlm.nih.gov (add the proper address prefix: http://www.) by searching the "proteins" section by use of the provided accession number. The content of this database is hereby incorporated by reference in its entirety.] In Figure 1F, The sequence of Influenza virus M2 is provided (PN0087/NCBI) (Seq. I.D. No. 12). In Figure 1H, For the Melanoma gp100 sequence (P40967/NCBI), substitute epitopes for agonists correspond to fldqvafsv (Seq. I.D. No. 1), fldqrvfvv (Seq. I.D. No. 2), and flflwffev (Seq. I.D. No. 3). In Figure 1H, alternate TRP-2 epitopes (CAA0437/NCI) are provided including llpggrpyr (Seq. I.D. No. 4) and lsvydffvw (Seq. I.D. No. 5). Figure 1L includes the following additional antigens: Prostate associated PAGE-1 (Homo sapiens) AAC25990/NCI (Seq. I.D. No. 34), Six

transmembrane epithelial antigen of the prostate (Homo sapiens) ACCESSION NP_036581/NCI (Seq. I.D. No. 35), G antigen, family C, 1; JM27 protein; prostate-associated gene protein 4 (Homo sapiens) - ACCESSION NP_008934 (Seq. I.D. No. 36), Mucin 1, transmembrane; (Homo sapiens) - ACCESSION NP_002447 (Seq. I.D. No. 37). Figure 1M includes the following additional antigens: Major surface glycoprotein G (Attachment glycoprotein G) of O. RSV - ACCESSION Q86695 (Seq. I.D. No. 38), Attachment glycoprotein (Human respiratory syncytial virus) - ACCESSION AAM82069 (Seq. I.D. No. 39), Glycoprotein G (Human herpes virus 1) - ACCESSION AAN04791 (Seq. I.D. No. 40), Immediate early protein ICP47 (human herpesvirus 1) - ACCESSION AAG33134 (Seq. I.D. No. 41), and Neurovirulence factor (ICP 34.5) (Human herpesvirus 2) - ACCESSION BAA23428 (Seq. I.D. No. 42);--

Please replace the paragraph starting on page 35, line 32 and ending on page 36, line 13, with the following paragraph:

--Figure 4(A) shows the detrimental effect of serum on the presentation of a T cell epitope peptide: M12 B cell lymphoma APC were incubated with TcH in the presence of various amounts of SFERFEIFPKE (HA) peptide (SEQ ID NO:5) in serum-free HL-1 medium ("HA+HL-1") or HL-1 medium supplemented with 20% mouse serum from BALB/c *scid* mice ("HA+serum"). The number of cells incubated was $2x10^4$M12 and $1x10^4$TcH/100µl of HL-1 medium supplemented or not with serum. The next day the plate was centrifuged for 15min/4^0C/1500RPM, then the supernatant was flicked, the cells were fixed with cold freshly made fixing solution (2% Formaldehyde, 0.2% Glutaraldehyde in 1X PBS) and the plate was again centrifuged for 3 min/4^0C/1500RPM. Fixing solution was flicked off the plate, cells washed once with PBS 200 µl/well, centrifuging the plate for 3min/4^0C/1500RPM. PBS was flicked off the plate and cells were incubated overnight at 37^0C with 200µl/well of the X-gal substrate freshly prepared as follows: 200µl of the X-gal stock solution, (40 mg/ml in DMSO) in 10ml of substrate buffer (5mM Potassium Ferrocyanide, 5mM Potassium Ferricyanide, 2mM $MgCl_2$ in 1X PBS). The blue activated TcH were scored visually using the microscope--

Please replace the paragraph starting on page 37, line 16 and ending on page 38, line 8, with the following paragraph:

--As shown in Figure 5A, *ex vivo* formation of MHC-peptide complexes on antigen presenting cells (APCs) from spleen was measured as follows: splenic APC were isolated by magnetic sorting using anti-MHC II antibodies. Separation by using magnetic beads coupled with anti-MHC II was carried out using magnetic cell separators and reagents from Miltenyi Biotec, Germany as follow: spleens were processed to single cell suspension, red blood cells lysed, then cells washed, counted and resuspended in MACS buffer (PBS supplemented with 2 mM EDTA and 0.5% BSA). Magnetically labeled cells were passed through a separation column which is placed in the magnetic field of a MACS separator. The magnetically labeled positive fraction is retained in the column while the negative fraction runs through. After removal of the column from the magnetic field, the magnetically retained positive cells are eluted from the column, cells are washed, counted, resuspended in HL1 complete media and they were incubated with specific T cell hybridoma recognizing I-E^d+ SFERFEIFPKE (SEQ ID NO:5) overnight, in the presence of various amounts of SFERFEIFPKE ("HA") peptide (SEQ ID NO:5) or recHA(I-Ed)-IgG ("IgHA"). Per well, $2x10^4$ APC were incubated with $1x10^4$ TcH. Next day the plate was centrifuged for 15min/4^0C/1500RPM, then the supernatant was flicked, the cells were fixed with cold freshly made fixing solution (2% Formaldehyde, 0.2% Glutaraldehyde in 1X PBS) and the plate was again centrifuged for 3min/4^0C/1500RPM. Fixing solution was flicked off the plate, cells washed once with PBS 200 µl/well, centrifuging the plate for 3min/4^0C/1500RPM. PBS was flicked off the plate and cells were incubated overnight at 37^0C with 200µl/well of the X-gal substrate freshly prepared as follows: 200µl of the X-gal stock solution, (40 mg/ml in DMSO) in 10ml of substrate buffer (5mM Potassium Ferrocyanide, 5mM Potassium Ferricyanide, 2mM $MgCl_2$ in 1X PBS). The blue activated TcH were scored visually using the microscope. The number of activated TcH was quantified and the results expressed as activation versus molar amount of epitope.--

Please replace the paragraph starting on page 39, line 17 and ending on page 39, line 29, with the following paragraph:

--(B) PBMC were used as APC with SFERFEIFPKE (HA)-specific TcH (SEQ ID NO:5), in the presence of cognate peptide or recHA (I-Ed)-IgG. The cells were co-incubated for 24 hours ($2x10^4$ APC + $1x10^4$ TcH). The next day the plate was centrifuged for 15min/4^0C/1500RPM, then the supernatant was flicked, the cells were fixed with cold freshly made fixing solution (2% Formaldehyde, 0.2% Glutaraldehyde in 1X PBS) and the plate was again centrifuged for 3min/4^0C/1500RPM. Fixing solution was flicked off the plate, cells washed once with PBS 200 µl/well, centrifuging the plate for 3min/4^0C/1500RPM. PBS was flicked off the plate and cells were incubated overnight at 37^0C with 200µl/well of the X-gal substrate freshly prepared as follows: 200µl of the X-gal stock solution, (40 mg/ml in DMSO) in 10ml of substrate buffer (5mM Potassium Ferrocyanide, 5mM Potassium Ferricyanide, 2mM $MgCl_2$ in 1X PBS). The blue activated TcH were scored visually using the microscope. The results are expressed as number of activated TcH /well, at different molar concentrations of epitope--

Please replace the paragraph starting on page 40, line 13 and ending on page 40, line 33, with the following paragraph:

--Assessment of *in vivo* formation of MHC-peptide complexes and a comparison with peptide in saline or standard oil-in-water emulsion were carried out in I-Ed^+ BALB/c mice. BALB/c mice were treated with recHA (I-Ed)-IgG, peptide in saline or peptide emulsified in incomplete Freund's adjuvant (IFA), by subcutaneous and intraperitoneal injection (doses depicted in Figure 7B). At 24 hours, the local (mesenteric) lymphoid nodes (LN), spleen and thymus were harvested, single cell suspensions were made, red blood cells lysed from the spleens, LN and thymus were collagenase digested. All cells were washed, counted and incubated with TcH recognizing I-ED+ SFERFEIFPKE (MHC class II-HA) (SEQ ID NO:5), complexes. The number of TcH was $1x10^4$/well. The formation of such MHC-peptide complexes was evaluated by titrating the number of APC with constant number of TcH and measuring TcH activation after overnight incubation. The next day the plate was centrifuged for

15min/4^{0}C/1500RPM, then the supernatant was flicked, the cells were fixed with cold freshly made fixing solution (2% Formaldehyde, 0.2% Glutaraldehyde in 1X PBS) and the plate was again centrifuged for 3min/4^{0}C/1500RPM. Fixing solution was flicked off the plate, cells washed once with PBS 200 µl/well, centrifuging the plate for 3min/4^{0}C/1500RPM: PBS was flicked off the plate and cells were incubated overnight at 37^{0}C with 200µl/well of the X-gal substrate freshly prepared as follows: 200µl of the X-gal stock solution, (40 mg/ml in DMSO) in 10ml of substrate buffer (5mM Potassium Ferrocyanide, 5mM Potassium Ferricyanide, 2mM $MgCl_2$ in 1X PBS). The blue activated TcH were scored visually using the microscope--

Please replace the paragraph starting on page 56, line 9 and ending on page 56, line 20, with the following paragraph:

--Activated SFERFEIFPKE-specific T cells (SEQ ID NO:5) were separated from BALB/c mice immunized 2 weeks previously with 100µg peptide in CFA. They were incubated with mitomycin treated splenocytes in the presence of various amounts of recHA(I-Ed)-IgG or corresponding peptide. The expansion and cytokine production (IFN-γ, IL-4, IL-2) was estimated by ELISPOT analysis as follows: the ELISPOT plates (Millipore, Molsheim, France) were incubated with purified anti-cytokine Abs (4µg/ml for anti-IL2 and anti-IL4, and 8 µg/ml for anti-IFN gamma, from BD Pharmingen) in sterile PBS (50 µg/well) at 4^{0}C overnight. The next day, the plates were washed 2 times with DMEM media and blocked with 200µl/well of DMEM complete containing FBS, for an hour, at 37^{0}C. Single cell suspension was made from the spleens, red blood cells were lysed, cells washed, counted and incubated at 5 x 10^5/well together with 20 µg/ml HA 110-120 peptide or just with media, to assess the background.--

AMENDMENTS TO THE SEQUENCE LISTING

Please insert the attached sequence listing, having pages numbered pages 1-35, into the specification after the abstract.

REMARKS/ARGUMENTS

Status of Claims

The specification has been amended to include specific sequence identifiers. Claim 1 has been amended to depend upon newly added claim 41 and to recite that the method is for inducing a Tc1 response in a patient. Claims 2-36 are cancelled. Claims 37-76 are added.

Support for New Claims

Support for new claims 37-76 can be found in the specification and claims as filed. For example, support for the recitation that the immunoglobulin is an IgG can be found at least at pages 27-32, support for the at least one T cell epitope can be found at least at pages 8, line 19; page 9, line 9; page 28, line 13; and original claims 1 and 10. Support for "covalently attached" can be found at least at page 3, line 1; and page 5, line 1. Support for "to the CDR region can be found at least at page 5, lines 8 and 9; and original claim 6, support for "modification of the Fc portion can be found at least at page 3, line 1; and page 5, lines 5 and 6. Support for the language "generates a Tc1 response in a patient" can be found at least at page 3, line 19. Support for the methods of treatment involving "virus immunization" or "treatment of tumor" can be found at least at pages 75, lines 26-28; and page 76, lines 11-14 using the claimed composition to vaccinate against infection challenge with a prototype virus; paragraphs 42-48 on pages 8 and 9 (virus immunization); examples 38-44, and paragraphs 49-58 on pages 9-10 (tumor treatment) using the claimed composition to treat tumors. As such, no new matter has been added herewith. As a result of the amendment claims 1 and 37-76 are presented for further examination.

Nucleotide and/or Amino Acid Sequences

In response to the Notice to Comply (box 7), Applicants have included specific sequence identifiers for Figures 1D, 1F, 1H, 1L, and 1M and pages 36-56. Enclosed herewith is a paper copy of the sequence listing as well as the required computer readable medium having the sequence listing.

Compliance with 37 C.F.R. §1.821-1.825 and the Sequence Listing

In order to comply with the Requirements for Patent Applications Containing Nucleotide Sequence and/or amino acid sequences disclosures, 37 C.F.R. §1.821-1.825, and further to the submission of a Sequence Listing, Applicants submit herewith the Sequence Listing in paper format, filed herewith, in compliance with 37 1.821(c) (which also complies with box 2 on the Notice to Comply). Further, the required computer readable medium having the sequence listing in compliance of 37 CFR 1.821(e) (which also complies with box 3 on the Notice To Comply).. As required under 37 C.F.R. §1.821(f) (and the Notice to Comply), I hereby verify that the data on the enclosed disk and paper copies of the Sequence Listing are identical. Pursuant to 37 C.F.R. §1.821(g) (and the Notice to comply), no new matter is being added herewith.

Rejection under 35 U.S.C.§112, first paragraph

Claims 1-36 were rejected under 35 U.S.C. §112, first paragraph, as not enabling for the method as claimed. In brief, the Office Action stated that "based on the state of the prior art, the breadth of the claims, and applicant's singe example of an NP-Ig capable of loading MHC class I and stimulating CD8+ T cells, and the single example of a combination of NP-Ig and pA:U dsRNA capable of stimulating cytotoxic T cells, it would have been considered unpredictable by the skilled artisan at the time of filing to use any antigenized immunoglobulin alone or in combination with pA:U to load MHC class I molecules on antigen presenting cells or to stimulate CD8+ T cells." See OA page 6, line 19 through page 7, line 2.

Claims 2-36 have been canceled. Claim 1 has been amended to be dependent upon the composition claim 41 and to specify that the agent used in the method of generating an enhanced T cell response is, the IgG having the at least one T cell epitope of the antigen covalently attached within the CDR region of the IgG without modification of the Fc portion and to recite that the method is for inducing a Tc1 response in a patient. The Tc1 response is a well understood and well-characterized immune response and is a cytotoxic immune response that involves high levels of IFNγ and IL2.

The test for enablement involves identifying whether the experimentation needed to practice the invention is undue or unreasonable (in re Wands, 858 F.2d 731, 737, 8 USPQ2d 1400, 1404 (Fed. Cir. 1988). There are many factors to be considered when determining whether

any experimentation needed is "undue." These include the breadth of the claims, the state of the prior art, the level of predictability in the art, the amount of direction provided, and the existence of working examples.

The currently claimed invention is enabled, because the specification as supplemented by the knowledge of the skilled artisan provides guidance as to how to use the combination of the recombinant IgG and dsRNA to induce a Tc1 T cell response. Neither the dsRNA nor the recombinant IgG were able by themselves to induce such a response. Further, as discussed below, appropriate balances of the Wands factors show the claimed invention is enabled:

With respect to the presence of working examples, Applicants respectfully submit that "nothing more than objective enablement is required, and therefore it is irrelevant whether this teaching is provided through broad terminology or illustrative examples." In re Wright, 999 F.2d 1557 (Fed. Cir. 1993).

Additionally, the Applicants disclose working examples of the use of the recombinant antibody and dsRNA of the claimed invention to activate Tc1 responses in viral and tumor applications in the specification on pages 75-83 (Examples 35-44). In addition to specific working examples, the specification provides direction to the skilled artisan on how to make, identify and use further conjugates as follows: there is teaching of the types of antigens (Figures 1E-1M and pp. 29-30) for which the claimed methods would be useful, there is teaching of the method of construction and production of the recombinant IgG (pp. 27-31), there is teaching of the purification of the recombinant IgG (pp. 31-32), there is teaching of the types of viral and tumor diseases that could be treated (pp. Figures 1E-1M and pp. 29-30), and there is teaching of the types of dsRNA that can be used in the combination (pp. 32-33). Thus, the data generated was relevant to both diseases, viral and cancer and was identified using a very well-characterized Tc epitope.

The teaching of the specification is supplemented by the knowledge of the skilled artisan, and the state of the prior art. Further, the predictability of the art is such that one of skill in the art could easily identify and produce claimed recombinant IgG and dsRNA combinations without undue experimentation. The specification provides exemplary recombinant IgG molecules and dsRNA molecules. There is a strong general knowledge of linkages and methods of producing such recombinant IgG molecules and dsRNA molecules. Also, the level of predictability for

producing recombinant IgG and dsRNA molecules for use in the methods is very high. Meaning that a working recombinant IgG and dsRNA combination can be made using the teaching in the specification with little uncertainty as to whether it will work. This is also true because the level of skill of the artisan is high, that of a Ph.D or experienced technician. Thus, Applicants respectfully submit that in view of the teachings of the specification, mentioned above, and the high level of ordinary skill in this art, undue experimentation would not be needed to make and use the claimed conjugates.

Rejection under 35 U.S.C.§112, second paragraph

Claims 13-29 were rejected under 35 U.S.C. §112, second paragraph, as being indefinite for the following reasons:

The Office Action stated that Claim 13 recites, "[a] method of loading an antigen presenting cells and generating an immune response to an antigen in a patient by use at least one peptide epitope attached to an immunoglobulin or portion thereof thereby forming an Ig-peptide complex wherein when the Ig-peptide complex is administered to a patient in vivo in conjunction with dsRNA...." (emphasis added by Office Action), but lacks any particular method steps.

In dependent claim 13 and dependent claims 14-29 have been canceled, rendering this rejection moot. Further, New claims 1, 68, 72 and 76 are drafted to contain specific method steps.

Claims 13-29 were further rejected under 35 U.S.C. §101 because the claimed recitation of a use, without setting forth any steps involved in the process, results in an improper definition of a process.

Independent claim 13 and dependent claims 14-29 have been canceled rendering this rejection moot. None of the new claims use the wording "for use..." or "wherein when..." or recite a use without reciting method steps.

Rejection under 35 U.S.C.§103(a)

The Office Action rejected claims 1-4, 6-7, 9-12, 30-32, and 34-36 under 35 U.S.C. §103(a) as unpatentable over US Patent No. 5,969,109 (1999), hereinafter referred to as Bona et

al. in view of US Patent No. 3,906,092 (1975), hereinafter referred to as Hilleman et al., and Cella et al. (1999) J. Exp. Med., Vol. 189(5), 821-829.

More specifically, the Office Action states that Bona et al. teaches methods of enhancing the cellular and humoral immune response to an antigen by administering a recombinant antibody comprising one or more viral T helper and/or B cell epitopes to a mammal and that the T and/or B cell epitopes are covalently attached to the immunoglobulin and further that the immunoglobulin was capable of stimulating HA specific CD4+ helper T cells. The Office Action further states that "Bona et al. differs from the present invention of failing to teach the co-administration of dsRNA with the antigenized immunoglobulin." But that "Hilleman et al. supplements Bona et al. by teaching that immune responses to antigen in adjuvant can be enhanced by the inclusion of dsRNA, such as poly A:U". While Hilleman et al. and Bona et al. do not teach that the dsRNA upregulates T cell response, the Office Action states that Cella et al. "supplements both Bona et al. and Hilleman et al. by teaching that dsRNA exhibits increased capacity to prime and polarize T cells."

The Claims

Claims 2-36 have been canceled. Claim 1 has been amended to be dependent upon the composition claim 41 and to specify that the agent used in the method of generating an enhanced T cell response is, "the IgG having the at least one T cell epitope of the antigen covalently attached within the CDR region of the IgG without modification of the Fc portion" and that the T cell response is "a Tc1 response." The Tc1 response is a well understood and well-characterised immune response and is a cytotoxic immune response that involves high levels of IFNγ and IL2

To establish a prima facie case of obviousness (MPEP 2143) states,

> First, there must be some suggestion or motivation, either in the references themselves or in the knowledge generally available to one of ordinary skill in the art, to modify the reference or to combine reference teachings. Second, there must be a reasonable expectation of success. Finally, the prior art reference (or references, when combined) must teach or suggest all the claim limitations. The teaching or suggestion to make the claimed combination and the reasonable expectation of success must both be found in the prior art, not in applicant's disclosure (*In re Vaeck*, 947 F.2d 488, 20 USPQ2d 1440 (Fed. Cir. 1991)).

Compare with: *Ortho-McNeil Pharmaceutical v. Mylan Labs* (Fed. Cir. 2008), 2007-1223, *Abbott Laboratories v. Sandoz, Inc.,* 544 F.3d 1341 (Fed. Cir. 2008), and *Proctor & Gamble Co. v Teva Pharm. USA, Inc.,* 2008-1404,-1405, -1406 (Fed. Cir. 2009).

In contrast to the requirements of MPEP 2143, it is shown that at the time of the invention, (1) the combination would not have taught all of the claimed limitations, (2) there was no motivation to combine, and (3) the resulting claimed invention would not have been expected by one or ordinary skill in the art.

The combination of references does not teach all of the claim limitations

The combination of Bona et al. Hilleman et al. and Cella et al. does not teach all of the claim elements, because neither Bona et al., Hilleman et al. nor Cella et al. teaches that the recombinant IgG and/or the dsRNA provides an enhanced Tc1 response. Bona et al, teaches that the recombinant antibodies can be used to induce an antibody response. Hilleman et al. is directed to use of polymers such as dsRNA as adjuvants in typical oil in water vaccines to increase the antibody response to the virus. Cella et al. is directed to the priming of T cells and specifically T helper responses.

The present inventors discovered that when recombinant antibodies are administered in conjunction with dsRNA, immunity was effectively redirected to the Tc1 response, thereby providing an enhanced Tc1 response. Claim 1 has been amended accordingly. Tc1 cells are a subpopulation of cytotoxic T cells characterized by secretion of IFNγ and IL2 but not IL4. Tc2 cells are a subpopulation of cytotoxic T cells characterized by secretion of IL4, but not IFNγ.

As discussed in the Applicant's specification, the Tc1 responses are associated with rearrangement of immunoglobulin genes to complement-dependent antibody, and the activation of phagocytic cells such as macrophages. Tc2 responses are associated with rearrangements of immunoglobulin genes to complement-independent and IgE antibodies and with the mobilization of nonphagocytic defenses such as eosinophils that combat microbes by the release of toxic factors. Tc1 responses are the desired response for fighting intracellular viral or bacterial infections, whereas Tc2 responses are favored for combating extracellular parasitic infection.

None of the references cited by the Office Action teaches induction of the specific subset of Tc1 responses. In fact, most of these references teach only an increased antibody response, having nothing to do with cytotoxic T cells. Antibody production is a humoral immune response, and while there are implications that T cells responses are included, these are only Thelper cells and are not implicated in the Tc response, which the presently claimed invention is concerned with. Even assuming that dsRNA was a known adjuvant, none of the references teach or suggest that when dsRNA is administered in combination with recombinant antibodies a Tc1 response is induced.

Thus, none of the cited art alone or in combination teaches that a combination of dsRNA with the IgG constructs would enhance the Tc1 response, and hence enable the viral and tumor treatment effects shown in the invention of claim 1.

Even if the combination did teach the induction of the Tc1 response, there would be no motivation to combine

Adjuvants have been used in the past to increase the antibody response to an antigen. This response includes both the humoral response (activation of B cells to produce antibodies), and the subset of the Tcell response that includes the activation of Thelper cells. Neither of these responses is part of the Tc1 response. Thus, the knowledge of the action of adjuvants would not be seen as a reason to add the dsRNA to a recombinant antibody to induce a Tc1 response and there would have been no motivation to combine the references to include the dsRNA with an IgG chimera instead of the claimed invention.

Further, it is known that inducing one arm of immunity (Th or B cell immunity) is not predictive for inducing another (Tc). The mechanism of induction of Tc (involving antigen processing through very specific pathways, presentation in the context of different molecules – MHCI to a distinct subset of T cells) is very different than that of Th or B cell immunity, and therefore at least without the teachings of the current specification, the Th or B cell immunity would not suggested to one of ordinary skill in the art the claimed mechanism of inducing Tc

There are unexpected properties

The combination of the recombinant IgG and the dsRNA as a whole possesses unexpected properties that could not have reasonably been viewed as a likely outcome of the invention. The wide range of possible outcomes and the relative unlikelihood that the resulting compound would exhibit the activation of the Tc1 response makes the method of using the agent and the dsRNA combination non-obvious. Neither the dsRNA nor the recombinant IgG were able by themselves to induce such a response. It was only when the two were administered in combination that the Tc1 response was possible.

Thus, in contrast to the requirements of MPEP 2143, at the time of the invention, (1) the combination would not have taught all of the claimed limitations because the specific Tc1 response was not taught, (2) there was no motivation to combine, because, if anything, that art suggested that including an IgG chimera instead of the claimed invention would have activated the humoral response rather than the Tc1 response, and (3) the resulting claimed invention would not have been expected by one or ordinary skill in the art, because only the previously undisclosed combination dsRNA recombinant IgG produces the response (neither produces the response alone).

CONCLUSION

In view of the foregoing, Applicants believe all claims now pending in this Application are in condition for allowance. The issuance of a formal Notice of Allowance at an early date is respectfully requested.

If the Examiner believes a telephone conference would expedite prosecution of this application, please telephone the undersigned at 408-993-1800.

Respectfully submitted,

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San Jose, CA 95110

SEQUENCE LISTING

<110> Bot, Adrian
Wang, Lilin
Smith, Dan
Phillips, Bill
Multicell Immunotherapeutics, Inc.

<120> Methods and Compositions to Generate and Control the Effector Profile of T Cells by Simultaneous Loading and Activation of Selected Subsets of Antigen Presenting Cells

<130> 8114-008-WO-US

<140> 10/527,931
<141> 2005-08-26

<150> US 60/412,219
<151> 2002-09-20

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        35   40   45
Val Asp Val Ser His Glu Asp Pro Glu Val Lys Phe Asn Trp Tyr Val
    50   55   60
Asp Gly Val Glu Val His Asn Ala Lys Thr Lys Pro Arg Glu Glu Gln
65   70   75   80
Tyr Asn Ser Thr Tyr Arg Val Val Ser Val Leu Thr Val Leu His Gln
                85   90   95
Asp Trp Leu Asn Gly Lys Glu Tyr Lys Cys Lys Val Phe Asn Lys Ala
            100   105   110
Leu Pro Ala Pro Ile Glu Lys Thr Ile Ser Lys Ala Lys Gly Gln Pro
        115   120   125
Arg Glu Pro Gln Val Tyr Thr Leu Pro Pro Ser Arg Glu Glu Met Thr
    130   135   140
Lys Asn Gln Val Ser Leu Thr Cys Leu Val Lys Gly Phe Tyr Pro Ser
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Asp Ile Ala Val Glu Trp Glu Ser Asn Gly Gln Pro Glu Asn Asn Tyr
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Lys Thr Thr Pro Pro Val Leu Asp Ser Asp Gly Ser Phe Phe Leu Tyr
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Ser Lys Leu Thr Val Asp Lys Ser Arg Trp Gln Gln Gly Asn Val Phe
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Ile Asp Tyr Glu Glu Leu Arg Glu Gln Leu Ser Ser Val Ser Ser Phe
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Glu Arg Phe Glu Ile Phe Pro Lys Glu Ser Ser Trp Pro Asn His Asn
    130                 135                 140
Thr Thr Lys Gly Val Thr Ala Ala Cys Ser His Ala Gly Lys Ser Ser
145                 150                 155                 160
Phe Tyr Arg Asn Leu Leu Trp Leu Thr Glu Lys Glu Gly Ser Tyr Pro
                165                 170                 175
Lys Leu Lys Asn Ser Tyr Val Asn Lys Lys Gly Lys Glu Val Leu Val
            180                 185                 190
Leu Trp Gly Ile His His Pro Ser Asn Ser Lys Asp Gln Gln Asn Ile
        195                 200                 205
Tyr Gln Asn Glu Asn Ala Tyr Val Ser Val Val Thr Ser Asn Tyr Asn
    210                 215                 220
Arg Arg Phe Thr Pro Glu Ile Ala Glu Arg Pro Lys Val Arg Asp Gln
225                 230                 235                 240
Ala Gly Arg Met Asn Tyr Tyr Trp Thr Leu Leu Lys Pro Gly Asp Thr
                245                 250                 255
Ile Ile Phe Glu Ala Asn Gly Asn Leu Ile Ala Pro Arg Tyr Ala Phe
            260                 265                 270
Ala Leu Ser Arg Gly Phe Gly Ser Gly Ile Ile Thr Ser Asn Ala Ser
        275                 280                 285
Met His Glu Cys Asn Thr Lys Cys Gln Thr Pro Leu Gly Ala Ile Asn
    290                 295                 300
Ser Ser Leu Pro Phe Gln Asn Ile His Pro Val Thr Ile Gly Glu Cys
305                 310                 315                 320
Pro Lys Tyr Val Arg Ser Ala Lys Leu Arg Met Val Thr Gly Leu Arg
                325                 330                 335
Asn Ile Pro Ser Ile Gln Ser Arg Gly Leu Phe Gly Ala Ile Ala Gly
            340                 345                 350
Phe Ile Glu Gly Gly Trp Thr Gly Met Ile Asp Gly Trp Tyr Gly Tyr
        355                 360                 365
His His Gln Asn Glu Gln Gly Ser Gly Tyr Ala Ala Asp Gln Lys Ser
    370                 375                 380
Thr Gln Asn Ala Ile Asn Gly Ile Thr Asn Lys Val Asn Ser Val Ile
385                 390                 395                 400
Glu Lys Met Asn Ile Gln Phe Thr Ala Val Gly Lys Glu Phe Asn Lys
                405                 410                 415
Leu Glu Lys Arg Met Glu Asn Leu Asn Lys Lys Val Asp Asp Gly Phe
            420                 425                 430
Leu Asp Ile Trp Thr Tyr Asn Ala Glu Leu Leu Val Leu Leu Glu Asn
        435                 440                 445
Glu Arg Thr Leu Asp Phe His Asp Ser Asn Val Lys Asn Leu Tyr Glu
    450                 455                 460
Lys Val Lys Ser Gln Leu Lys Asn Asn Ala Lys Glu Ile Gly Asn Gly
465                 470                 475                 480
Cys Phe Glu Phe Tyr His Lys Cys Asp Asn Glu Cys Met Glu Ser Val
                485                 490                 495
```

```
Arg Asn Gly Thr Tyr Asp Tyr Pro Lys Tyr Ser Glu Glu Ser Lys Leu
            500                 505                 510
Asn Arg Glu Lys Val Asp Gly Val Lys Leu Glu Ser Met Gly Ile Tyr
        515                 520                 525
Gln Ile Leu Ala Ile Tyr Ser Thr Val Ala Ser Ser Leu Val Leu Leu
    530                 535                 540
Val Ser Leu Gly Ala Ile Ser Phe Trp Met Cys Ser Asn Gly Ser Leu
545                 550                 555                 560
Gln Cys Arg Ile Cys Ile
                565
```

<210> 9
<211> 498
<212> PRT
<213> Influenza A virus

<220>
<223> influenza virus A/Puerto Rico/8/34 nucleoprotein (NP)

<400> 9

```
Met Ala Ser Gln Gly Thr Lys Arg Ser Tyr Glu Gln Met Glu Thr Gly
 1               5                  10                  15
Gly Glu Arg Gln Asn Ala Thr Glu Ile Arg Ala Ser Val Gly Arg Met
            20                  25                  30
Val Gly Gly Ile Gly Arg Phe Tyr Ile Gln Met Cys Thr Glu Leu Gln
        35                  40                  45
Leu Ser Asp Tyr Glu Gly Arg Leu Ile Gln Asn Ser Ile Thr Ile Glu
    50                  55                  60
Arg Met Val Leu Ser Ala Phe Asp Glu Arg Arg Asn Lys Tyr Leu Glu
65                  70                  75                  80
Glu His Pro Ser Ala Gly Lys Asp Pro Lys Lys Thr Gly Gly Pro Ile
                85                  90                  95
Tyr Lys Lys Arg Asp Gly Lys Trp Met Arg Glu Leu Ile Leu Tyr Asp
            100                 105                 110
Lys Asp Glu Ile Arg Arg Ile Trp Arg Gln Ala Asn Asn Gly Glu Asp
        115                 120                 125
Ala Thr Ala Gly Leu Thr His Leu Met Ile Trp His Ser Asn Leu Asn
    130                 135                 140
Asp Ala Thr Tyr Gln Arg Thr Arg Ala Leu Val Arg Thr Gly Met Asp
145                 150                 155                 160
Pro Arg Met Cys Ser Leu Met Gln Gly Ser Thr Leu Pro Arg Arg Ser
                165                 170                 175
Gly Ala Ala Gly Ala Ala Val Lys Gly Ile Gly Thr Met Val Met Glu
            180                 185                 190
Leu Ile Arg Met Ile Lys Arg Gly Ile Asn Asp Arg Asn Phe Trp Arg
        195                 200                 205
Gly Glu Asn Gly Arg Arg Thr Arg Ile Ala Tyr Glu Arg Met Cys Asn
    210                 215                 220
Ile Leu Lys Gly Lys Phe Gln Thr Ala Ala Gln Arg Ala Met Met Asp
225                 230                 235                 240
Gln Val Arg Glu Ser Arg Asn Pro Gly Asn Ala Glu Ile Glu Asp Leu
                245                 250                 255
```

```
Ile Phe Leu Ala Arg Ser Ala Leu Ile Leu Arg Gly Ser Val Ala His
            260                 265                 270
Lys Ser Cys Leu Pro Ala Cys Ile Tyr Gly Leu Val Val Ala Ser Gly
        275                 280                 285
Tyr Asp Phe Glu Arg Glu Gly Tyr Ser Leu Val Gly Ile Asp Pro Phe
    290                 295                 300
Arg Leu Leu Gln Asn Ser Gln Val Phe Ser Leu Ile Arg Pro Asn Glu
305                 310                 315                 320
Asn Pro Val His Lys Ser Gln Leu Ile Trp Met Ala Cys His Ser Ala
                325                 330                 335
Ala Phe Glu Asp Leu Arg Val Ser Ser Phe Ile Arg Gly Thr Lys Val
            340                 345                 350
Val Pro Arg Gly Gln Leu Thr Thr Arg Gly Val Gln Ile Ala Ser Asn
        355                 360                 365
Glu Asn Met Glu Thr Met Asp Ser Ile Thr Leu Glu Leu Arg Ser Lys
    370                 375                 380
Tyr Trp Ala Ile Arg Thr Arg Ser Gly Gly Asn Thr Asn Gln Gln Arg
385                 390                 395                 400
Ala Ser Ala Gly Gln Ile Ser Val Gln Pro Thr Phe Ser Val Gln Arg
                405                 410                 415
Asn Leu Pro Phe Glu Arg Ala Thr Ile Met Ala Ala Phe Thr Gly Asn
            420                 425                 430
Asn Glu Gly Arg Thr Ser Asp Met Arg Thr Glu Ile Ile Arg Met Met
        435                 440                 445
Glu Ser Ala Arg Pro Asp Asp Val Ser Phe Gln Gly Arg Gly Val Phe
    450                 455                 460
Glu Leu Ser Asp Glu Lys Ala Thr Asn Pro Ile Val Pro Ser Phe Asp
465                 470                 475                 480
Met Ser Asn Glu Gly Ser Tyr Phe Phe Gly Asp Asn Ala Glu Glu Tyr
                485                 490                 495
Asp Asn
```

<210> 10
<211> 114
<212> PRT
<213> Artificial Sequence

<220>
<223> murine anti-arsonate antibody G1MSAA VH segment

<400> 10

```
Glu Val Gln Leu Gln Gln Ser Gly Ala Glu Leu Val Lys Ala Gly Ser
 1               5                  10                  15
Ser Val Lys Met Ser Cys Lys Ala Thr Gly Tyr Thr Phe Ser Ser Tyr
            20                  25                  30
Glu Leu Tyr Trp Val Arg Gln Ala Pro Gly Gln Gly Leu Glu Asp Leu
        35                  40                  45
Gly Tyr Ile Ser Ser Ser Ser Ala Tyr Pro Asn Tyr Ala Gln Lys Phe
    50                  55                  60
Gln Gly Arg Val Thr Ile Thr Ala Asp Glu Ser Thr Asn Thr Ala Tyr
65                  70                  75                  80
Met Glu Leu Ser Ser Leu Arg Ser Glu Asp Thr Ala Val Tyr Phe Cys
                85                  90                  95
```

```
Ala Val Arg Val Ile Ser Arg Tyr Phe Asp Gly Trp Gly Gln Gly Thr
            100                 105                 110
Leu Val


<210> 11
<211> 252
<212> PRT
<213> Influenza A virus

<220>
<223> Influenza A virus matrix protein M1

<400> 11
Met Ser Leu Leu Thr Glu Val Glu Thr Tyr Val Leu Ser Ile Ile Pro
 1               5                  10                  15
Ser Gly Pro Leu Lys Ala Glu Ile Ala Gln Arg Leu Glu Asp Val Phe
            20                  25                  30
Ala Gly Lys Asn Thr Asp Leu Glu Val Leu Met Glu Trp Leu Lys Thr
        35                  40                  45
Arg Pro Ile Leu Ser Pro Leu Thr Lys Gly Val Leu Gly Phe Val Phe
    50                  55                  60
Thr Leu Thr Val Pro Ser Glu Arg Gly Leu Gln Arg Arg Arg Phe Val
65                  70                  75                  80
Gln Asn Ala Leu Asn Gly Asn Gly Asp Pro Asn Asn Met Asp Lys Ala
                85                  90                  95
Val Lys Leu Tyr Arg Lys Leu Lys Arg Glu Ile Thr Phe Tyr Gly Ala
            100                 105                 110
Lys Glu Val Ala Leu Ser Tyr Ser Thr Gly Ala Leu Ala Ser Cys Met
        115                 120                 125
Gly Leu Ile Tyr Asn Arg Met Gly Thr Val Thr Thr Glu Val Ala Phe
    130                 135                 140
Gly Leu Val Cys Ala Thr Cys Glu Gln Ile Ala Asp Ser Gln His Arg
145                 150                 155                 160
Ser His Arg Gln Met Val Thr Thr Thr Asn Pro Leu Ile Arg His Glu
                165                 170                 175
Asn Arg Met Val Leu Ala Ser Thr Thr Ala Lys Ala Met Glu Gln Met
            180                 185                 190
Ala Gly Ser Ser Glu Gln Ala Ala Glu Ala Met Glu Val Ala Ser Gln
        195                 200                 205
Ala Arg Gln Met Val Gln Ala Met Arg Thr Val Gly Thr His Pro Ser
    210                 215                 220
Ser Ser Ala Gly Leu Lys Asp Asp Leu Leu Glu Asn Leu Gln Ala Tyr
225                 230                 235                 240
Gln Lys Arg Met Gly Val Gln Leu Gln Arg Phe Lys
                245                 250


<210> 12
<211> 97
<212> PRT
<213> Influenza A virus

<220>
```

<223> Influenza A virus matrix protein M2

<400> 12
Met Ser Leu Leu Thr Glu Val Glu Thr Pro Thr Arg Asn Gly Trp Glu
 1   5   10   15
Cys Ser Cys Ser Asp Ser Ser Asp Pro Leu Val Ile Ala Ala Ser Ile
            20   25   30
Ile Gly Ile Leu His Phe Ile Leu Trp Ile Leu Asp Arg Leu Phe Phe
        35   40   45
Lys Cys Ile Tyr Arg Arg Leu Lys Tyr Gly Leu Lys Arg Gly Pro Ser
    50   55   60
Thr Glu Gly Val Pro Lys Ser Met Arg Glu Glu Tyr Arg Gln Glu Gln
65   70   75   80
Gln Asn Ala Val Asp Val Asp Asp Gly His Phe Val Asn Ile Glu Leu
                85   90   95
Glu

<210> 13
<211> 181
<212> PRT
<213> Hepatitis C virus

<220>
<223> Hepatitis C virus (HCV) NS3 protease

<400> 13
Ala Pro Ile Thr Ala Tyr Ala Gln Gln Thr Arg Gly Leu Leu Gly Cys
 1   5   10   15
Ile Ile Thr Ser Leu Thr Gly Arg Asp Arg Asn Gln Val Glu Gly Glu
            20   25   30
Val Gln Val Val Ser Thr Ala Thr Gln Ser Phe Leu Ala Thr Cys Ile
        35   40   45
Asn Gly Val Cys Trp Thr Val Phe His Gly Ala Gly Ser Lys Thr Leu
    50   55   60
Ala Gly Pro Lys Gly Pro Ile Thr Gln Met Tyr Thr Asn Val Asp Gln
65   70   75   80
Asp Leu Val Gly Trp Pro Ala Pro Pro Gly Ala Arg Ser Leu Thr Pro
                85   90   95
Cys Thr Cys Gly Ser Ser Asp Leu Tyr Leu Val Thr Arg His Ala Asp
            100   105   110
Val Val Pro Val Arg Arg Arg Ser Asp Ser Arg Gly Ser Leu Leu Ser
        115   120   125
Pro Arg Pro Ile Ser Tyr Leu Lys Gly Ser Ser Gly Gly Pro Leu Leu
    130   135   140
Cys Pro Ser Gly His Ala Val Gly Ile Phe Arg Ala Ala Val Cys Thr
145   150   155   160
Arg Gly Val Ala Lys Ala Val Asp Phe Val Pro Val Glu Ser Met Glu
                165   170   175
Thr Thr Met Arg Ser
            180

<210> 14

<211> 139
<212> PRT
<213> Hepatitis B virus

<220>
<223> Hepatitis B virus (HBV) core antigen

<400> 14
Met Asp Ile Asp Pro Tyr Lys Glu Phe Gly Ala Ser Val Glu Leu Leu
 1   5   10   15
Ser Phe Leu Pro Ser Asp Phe Phe Pro Ser Ile Arg Asp Leu Leu Asp
            20   25   30
Thr Ala Ser Ala Leu Tyr Arg Glu Ala Leu Glu Ser Pro Glu His Cys
        35   40   45
Ser Pro His His Thr Ala Leu Arg Gln Ala Ile Leu Cys Trp Gly Glu
    50   55   60
Leu Met Asn Leu Ala Thr Trp Val Gly Ser Asn Leu Glu Asp Pro Ala
65   70   75   80
Ser Arg Glu Leu Val Val Ser Tyr Val Asn Val Asn Met Gly Leu Lys
                85   90   95
Ile Arg Gln Leu Leu Arg Phe His Ile Ser Cys Leu Thr Phe Gly Arg
            100   105   110
Glu Thr Val Leu Glu Tyr Leu Val Ser Phe Gly Val Trp Ile Arg Thr
        115   120   125
Pro Pro Ala Tyr Arg Pro Pro Asn Ala Pro Ile
    130   135

<210> 15
<211> 105
<212> PRT
<213> Human papilloma virus type 18

<220>
<223> Human papilloma virus type 18 (HPV-18) E7 protein

<400> 15
Met His Gly Pro Lys Ala Thr Leu Gln Asp Ile Val Leu His Leu Glu
 1   5   10   15
Pro Gln Asn Glu Ile Pro Val Asp Leu Leu Cys His Glu Gln Leu Ser
            20   25   30
Asp Ser Glu Glu Glu Asn Asp Glu Ile Asp Gly Val Asn His Gln His
        35   40   45
Leu Pro Ala Arg Arg Ala Glu Pro Gln Arg His Thr Met Leu Cys Met
    50   55   60
Cys Cys Lys Cys Glu Ala Arg Ile Lys Leu Val Val Glu Ser Ser Ala
65   70   75   80
Asp Asp Leu Arg Ala Phe Gln Gln Leu Phe Leu Asn Thr Leu Ser Phe
                85   90   95
Val Cys Pro Trp Cys Ala Ser Gln Gln
            100   105

<210> 16

<211> 98
<212> PRT
<213> Human papilloma virus type 16

<220>
<223> Human papilloma virus type 16 (HPV-16) E7 protein

<400> 16
Met His Gly Asp Thr Pro Thr Leu His Glu Tyr Met Leu Asp Leu Gln
 1   5   10   15
Pro Glu Thr Thr Asp Leu Tyr Cys Tyr Glu Gln Leu Asn Asp Ser Ser
            20   25   30
Glu Glu Glu Asp Glu Ile Asp Gly Pro Ala Gly Gln Ala Glu Pro Asp
        35   40   45
Arg Ala His Tyr Asn Ile Val Thr Phe Cys Cys Lys Cys Asp Ser Thr
    50   55   60
Leu Arg Leu Cys Val Gln Ser Thr His Val Asp Ile Arg Thr Leu Glu
65   70   75   80
Asp Leu Leu Met Gly Thr Leu Gly Ile Val Cys Pro Ile Cys Ser Gln
                85   90   95
Lys Pro

<210> 17
<211> 158
<212> PRT
<213> Human papilloma virus type 18

<220>
<223> Human papilloma virus type 18 (HPV-18) E6 protein

<400> 17
Met Ala Arg Phe Glu Asp Pro Thr Arg Arg Pro Tyr Lys Leu Pro Asp
 1   5   10   15
Leu Cys Thr Glu Leu Asn Thr Ser Leu Gln Asp Ile Glu Ile Thr Cys
            20   25   30
Val Tyr Cys Lys Thr Val Leu Glu Leu Thr Glu Val Phe Glu Phe Ala
        35   40   45
Phe Lys Asp Leu Phe Val Val Tyr Arg Asp Ser Ile Pro His Ala Ala
    50   55   60
Cys His Lys Cys Ile Asp Phe Tyr Ser Arg Ile Arg Glu Leu Arg His
65   70   75   80
Tyr Ser Asp Ser Val Tyr Gly Asp Thr Leu Glu Lys Leu Thr Asn Thr
                85   90   95
Gly Leu Tyr Asn Leu Leu Ile Arg Cys Leu Arg Cys Gln Lys Pro Leu
            100   105   110
Asn Pro Ala Glu Lys Leu Arg His Leu Asn Glu Lys Arg Arg Phe His
        115   120   125
Lys Ile Ala Gly His Tyr Arg Gly Gln Cys His Ser Cys Cys Asn Arg
    130   135   140
Ala Arg Gln Glu Arg Leu Gln Arg Arg Arg Glu Thr Gln Val
145   150   155

<210> 18
<211> 158
<212> PRT
<213> Human papilloma virus type 16

<220>
<223> Human papilloma virus type 16 (HPV-16) E6 protein

<400> 18
Met His Gln Lys Arg Thr Ala Met Phe Gln Asp Pro Gln Glu Arg Pro
 1   5   10   15
Arg Lys Leu Pro His Leu Cys Thr Glu Leu Gln Thr Thr Ile His Asp
            20   25   30
Ile Ile Leu Glu Cys Val Tyr Cys Lys Gln Gln Leu Leu Arg Arg Glu
        35   40   45
Val Tyr Asp Phe Ala Phe Arg Asp Leu Cys Ile Val Tyr Arg Asp Gly
    50   55   60
Asn Pro Tyr Ala Val Cys Asp Lys Cys Leu Lys Phe Tyr Ser Lys Ile
65   70   75   80
Ser Glu Tyr Arg Tyr Tyr Cys Tyr Ser Val Tyr Gly Thr Thr Leu Glu
                85   90   95
Gln Gln Tyr Asn Lys Pro Leu Cys Asp Leu Leu Ile Arg Cys Ile Asn
            100   105   110
Cys Gln Lys Pro Leu Cys Pro Glu Glu Lys Gln Arg His Leu Asp Lys
        115   120   125
Lys Gln Arg Phe His Asn Ile Arg Gly Arg Trp Thr Gly Arg Cys Met
    130   135   140
Ser Cys Cys Arg Ser Ser Arg Thr Arg Arg Glu Thr Gln Leu
145   150   155

<210> 19
<211> 661
<212> PRT
<213> Homo sapiens

<220>
<223> melanocyte lineage-specific antigen gp100, melanocyte
      protein Pmel 17 (Silver locus protein homolog, SILV),
      melanoma-associated ME20 antigen (ME20-M), 95 kDa
      melanocyte-specific secreted glycoprotein

<400> 19
Met Asp Leu Val Leu Lys Arg Cys Leu Leu His Leu Ala Val Ile Gly
 1   5   10   15
Ala Leu Leu Ala Val Gly Ala Thr Lys Val Pro Arg Asn Gln Asp Trp
            20   25   30
Leu Gly Val Ser Arg Gln Leu Arg Thr Lys Ala Trp Asn Arg Gln Leu
        35   40   45
Tyr Pro Glu Trp Thr Glu Ala Gln Arg Leu Asp Cys Trp Arg Gly Gly
    50   55   60
Gln Val Ser Leu Lys Val Ser Asn Asp Gly Pro Thr Leu Ile Gly Ala
65   70   75   80
Asn Ala Ser Phe Ser Ile Ala Leu Asn Phe Pro Gly Ser Gln Lys Val

```
                85                  90                  95
Leu Pro Asp Gly Gln Val Ile Trp Val Asn Asn Thr Ile Ile Asn Gly
            100                 105                 110
Ser Gln Val Trp Gly Gly Gln Pro Val Tyr Pro Gln Glu Thr Asp Asp
        115                 120                 125
Ala Cys Ile Phe Pro Asp Gly Gly Pro Cys Pro Ser Gly Ser Trp Ser
    130                 135                 140
Gln Lys Arg Ser Phe Val Tyr Val Trp Lys Thr Trp Gly Gln Tyr Trp
145                 150                 155                 160
Gln Val Leu Gly Gly Pro Val Ser Gly Leu Ser Ile Gly Thr Gly Arg
                165                 170                 175
Ala Met Leu Gly Thr His Thr Met Glu Val Thr Val Tyr His Arg Arg
            180                 185                 190
Gly Ser Arg Ser Tyr Val Pro Leu Ala His Ser Ser Ser Ala Phe Thr
        195                 200                 205
Ile Thr Asp Gln Val Pro Phe Ser Val Ser Val Ser Gln Leu Arg Ala
    210                 215                 220
Leu Asp Gly Gly Asn Lys His Phe Leu Arg Asn Gln Pro Leu Thr Phe
225                 230                 235                 240
Ala Leu Gln Leu His Asp Pro Ser Gly Tyr Leu Ala Glu Ala Asp Leu
                245                 250                 255
Ser Tyr Thr Trp Asp Phe Gly Asp Ser Ser Gly Thr Leu Ile Ser Arg
            260                 265                 270
Ala Leu Val Val Thr His Thr Tyr Leu Glu Pro Gly Pro Val Thr Ala
        275                 280                 285
Gln Val Val Leu Gln Ala Ala Ile Pro Leu Thr Ser Cys Gly Ser Ser
    290                 295                 300
Pro Val Pro Gly Thr Thr Asp Gly His Arg Pro Thr Ala Glu Ala Pro
305                 310                 315                 320
Asn Thr Thr Ala Gly Gln Val Pro Thr Thr Glu Val Val Gly Thr Thr
                325                 330                 335
Pro Gly Gln Ala Pro Thr Ala Glu Pro Ser Gly Thr Thr Ser Val Gln
            340                 345                 350
Val Pro Thr Thr Glu Val Ile Ser Thr Ala Pro Val Gln Met Pro Thr
        355                 360                 365
Ala Glu Ser Thr Gly Met Thr Pro Glu Lys Val Pro Val Ser Glu Val
    370                 375                 380
Met Gly Thr Thr Leu Ala Glu Met Ser Thr Pro Glu Ala Thr Gly Met
385                 390                 395                 400
Thr Pro Ala Glu Val Ser Ile Val Val Leu Ser Gly Thr Thr Ala Ala
                405                 410                 415
Gln Val Thr Thr Thr Glu Trp Val Glu Thr Thr Ala Arg Glu Leu Pro
            420                 425                 430
Ile Pro Glu Pro Glu Gly Pro Asp Ala Ser Ser Ile Met Ser Thr Glu
        435                 440                 445
Ser Ile Thr Gly Ser Leu Gly Pro Leu Leu Asp Gly Thr Ala Thr Leu
    450                 455                 460
Arg Leu Val Lys Arg Gln Val Pro Leu Asp Cys Val Leu Tyr Arg Tyr
465                 470                 475                 480
Gly Ser Phe Ser Val Thr Leu Asp Ile Val Gln Gly Ile Glu Ser Ala
                485                 490                 495
Glu Ile Leu Gln Ala Val Pro Ser Gly Glu Gly Asp Ala Phe Glu Leu
            500                 505                 510
Thr Val Ser Cys Gln Gly Gly Leu Pro Lys Glu Ala Cys Met Glu Ile
```

```
        515                 520                 525
Ser Ser Pro Gly Cys Gln Pro Pro Ala Gln Arg Leu Cys Gln Pro Val
    530                 535                 540
Leu Pro Ser Pro Ala Cys Gln Leu Val Leu His Gln Ile Leu Lys Gly
545                 550                 555                 560
Gly Ser Gly Thr Tyr Cys Leu Asn Val Ser Leu Ala Asp Thr Asn Ser
                565                 570                 575
Leu Ala Val Val Ser Thr Gln Leu Ile Met Pro Gly Gln Glu Ala Gly
            580                 585                 590
Leu Gly Gln Val Pro Leu Ile Val Gly Ile Leu Leu Val Leu Met Ala
        595                 600                 605
Val Val Leu Ala Ser Leu Ile Tyr Arg Arg Arg Leu Met Lys Gln Asp
    610                 615                 620
Phe Ser Val Pro Gln Leu Pro His Ser Ser Ser His Trp Leu Arg Leu
625                 630                 635                 640
Pro Arg Ile Phe Cys Ser Cys Pro Ile Gly Glu Asn Ser Pro Leu Leu
                645                 650                 655
Ser Gly Gln Gln Val
            660
```

<210> 20
<211> 118
<212> PRT
<213> Homo sapiens

<220>
<223> melanoma antigen recognized by T-cells 1 (MART-1, MLANA), Melan-A protein, antigen SK29-AA, antigen LB39-AA

<400> 20

```
Met Pro Arg Glu Asp Ala His Phe Ile Tyr Gly Tyr Pro Lys Lys Gly
 1               5                  10                  15
His Gly His Ser Tyr Thr Thr Ala Glu Glu Ala Ala Gly Ile Gly Ile
            20                  25                  30
Leu Thr Val Ile Leu Gly Val Leu Leu Leu Ile Gly Cys Trp Tyr Cys
        35                  40                  45
Arg Arg Arg Asn Gly Tyr Arg Ala Leu Met Asp Lys Ser Leu His Val
    50                  55                  60
Gly Thr Gln Cys Ala Leu Thr Arg Arg Cys Pro Gln Glu Gly Phe Asp
65                  70                  75                  80
His Arg Asp Ser Lys Val Ser Leu Gln Glu Lys Asn Cys Glu Pro Val
                85                  90                  95
Val Pro Asn Ala Pro Pro Ala Tyr Glu Lys Leu Ser Ala Glu Gln Ser
            100                 105                 110
Pro Pro Pro Tyr Ser Pro
        115
```

<210> 21
<211> 237
<212> PRT
<213> Homo sapiens

<220>
<223> tyrosinase-related protein-2 (TRP-2, TYRP2),
DOPAchrome tautomerase

<400> 21
Met Ser Pro Leu Trp Trp Gly Phe Leu Leu Ser Cys Leu Gly Cys Lys
1 5 10 15
Ile Leu Pro Gly Ala Gln Gly Gln Phe Pro Arg Val Cys Met Thr Val
20 25 30
Asp Ser Leu Val Asn Lys Glu Cys Cys Pro Arg Leu Gly Ala Glu Ser
35 40 45
Ala Asn Val Cys Gly Ser Gln Gln Gly Arg Gly Gln Cys Thr Glu Val
50 55 60
Arg Ala Asp Thr Arg Pro Trp Ser Gly Pro Tyr Ile Leu Arg Asn Gln
65 70 75 80
Asp Asp Arg Glu Leu Trp Pro Arg Lys Phe Phe His Arg Thr Cys Lys
85 90 95
Cys Thr Gly Asn Phe Ala Gly Tyr Asn Cys Gly Asp Cys Lys Phe Gly
100 105 110
Trp Thr Gly Pro Asn Cys Glu Arg Lys Lys Pro Pro Val Ile Arg Gln
115 120 125
Asn Ile His Ser Leu Ser Pro Gln Glu Arg Glu Gln Phe Leu Gly Ala
130 135 140
Leu Asp Leu Ala Lys Lys Arg Val His Pro Asp Tyr Val Ile Thr Thr
145 150 155 160
Gln His Trp Val Gly Leu Leu Gly Pro Asn Gly Thr Gln Pro Gln Phe
165 170 175
Ala Asn Cys Ser Val Tyr Asp Phe Phe Val Trp Leu His Tyr Tyr Ser
180 185 190
Val Arg Asp Thr Leu Leu Gly Gly Phe Phe Pro Trp Leu Lys Val Tyr
195 200 205
Tyr Tyr Arg Phe Val Ile Gly Leu Arg Val Trp Gln Trp Glu Val Ile
210 215 220
Ser Cys Lys Leu Ile Lys Arg Ala Thr Thr Arg Gln Pro
225 230 235

<210> 22
<211> 337
<212> PRT
<213> Homo sapiens

<220>
<223> carcinoma embryonic antigen precursor,
carcinoembryonic antigen-related cell adhesion
molecule 18

<400> 22
Met Asp Leu Ser Arg Pro Arg Trp Ser Leu Trp Arg Arg Val Phe Leu
1 5 10 15
Met Ala Ser Leu Leu Ala Cys Gly Ile Cys Gln Ala Ser Gly Gln Ile
20 25 30
Phe Ile Thr Gln Thr Leu Gly Ile Lys Gly Tyr Arg Thr Val Val Ala

```
        35                  40                  45
Leu Asp Lys Val Pro Glu Asp Val Gln Glu Tyr Ser Trp Tyr Trp Gly
    50                  55                  60
Ala Asn Asp Ser Ala Gly Asn Met Ile Ile Ser His Lys Pro Pro Ser
65                  70                  75                  80
Ala Gln Gln Pro Gly Pro Met Tyr Thr Gly Arg Glu Arg Val Asn Arg
                85                  90                  95
Glu Gly Ser Leu Leu Ile Arg Pro Thr Ala Leu Asn Asp Thr Gly Asn
            100                 105                 110
Tyr Thr Val Arg Val Val Ala Gly Asn Glu Thr Gln Arg Ala Thr Gly
        115                 120                 125
Trp Leu Glu Val Leu Glu Leu Gly Ser Asn Leu Gly Ile Ser Val Asn
    130                 135                 140
Ala Ser Ser Leu Val Glu Asn Met Asp Ser Val Ala Ala Asp Cys Leu
145                 150                 155                 160
Thr Asn Val Thr Asn Ile Thr Trp Tyr Val Asn Asp Val Pro Thr Ser
                165                 170                 175
Ser Ser Asp Arg Met Thr Ile Ser Pro Asp Gly Lys Thr Leu Val Ile
            180                 185                 190
Leu Arg Val Ser Arg Tyr Asp Arg Thr Ile Gln Cys Met Ile Glu Ser
        195                 200                 205
Phe Pro Glu Ile Phe Gln Arg Ser Glu Arg Ile Ser Leu Thr Val Ala
    210                 215                 220
Tyr Gly Pro Asp Tyr Val Leu Leu Arg Ser Asn Pro Asp Asp Phe Asn
225                 230                 235                 240
Gly Ile Val Thr Ala Glu Ile Gly Ser Gln Val Glu Met Glu Cys Ile
                245                 250                 255
Cys Tyr Ser Phe Leu Asp Leu Lys Tyr His Trp Ile His Asn Gly Ser
            260                 265                 270
Leu Leu Asn Phe Ser Asp Ala Lys Met Asn Leu Ser Ser Leu Ala Trp
        275                 280                 285
Glu Gln Met Gly Arg Tyr Arg Cys Thr Val Glu Asn Pro Val Thr Gln
    290                 295                 300
Leu Ile Met Tyr Met Asp Val Arg Ile Gln Ala Pro His Glu Cys Ser
305                 310                 315                 320
Ser Ser Pro Pro Gly Ser Cys Phe Ala His Leu Pro Ala Ser Met Pro
                325                 330                 335
Cys
```

<210> 23
<211> 1255
<212> PRT
<213> Homo sapiens

<220>
<223> Her-2/neu protein, tyrosine kinase-type cell surface receptor Her-2, v-erb-b2 erythroblastic leukemia viral oncogene-like 2 (erbB-2), herstatin, neuro/glioblastoma derived oncogene homolog, receptor tyrosine-protein kinase erbB-2 (p185erbB2)

<400> 23

```
Met Glu Leu Ala Ala Leu Cys Arg Trp Gly Leu Leu Leu Ala Leu Leu
 1               5                  10                  15
```

```
Pro Pro Gly Ala Ala Ser Thr Gln Val Cys Thr Gly Thr Asp Met Lys
            20                  25                  30
Leu Arg Leu Pro Ala Ser Pro Glu Thr His Leu Asp Met Leu Arg His
        35                  40                  45
Leu Tyr Gln Gly Cys Gln Val Val Gln Gly Asn Leu Glu Leu Thr Tyr
    50                  55                  60
Leu Pro Thr Asn Ala Ser Leu Ser Phe Leu Gln Asp Ile Gln Glu Val
65                  70                  75                  80
Gln Gly Tyr Val Leu Ile Ala His Asn Gln Val Arg Gln Val Pro Leu
                85                  90                  95
Gln Arg Leu Arg Ile Val Arg Gly Thr Gln Leu Phe Glu Asp Asn Tyr
            100                 105                 110
Ala Leu Ala Val Leu Asp Asn Gly Asp Pro Leu Asn Asn Thr Thr Pro
        115                 120                 125
Val Thr Gly Ala Ser Pro Gly Gly Leu Arg Glu Leu Gln Leu Arg Ser
    130                 135                 140
Leu Thr Glu Ile Leu Lys Gly Gly Val Leu Ile Gln Arg Asn Pro Gln
145                 150                 155                 160
Leu Cys Tyr Gln Asp Thr Ile Leu Trp Lys Asp Ile Phe His Lys Asn
                165                 170                 175
Asn Gln Leu Ala Leu Thr Leu Ile Asp Thr Asn Arg Ser Arg Ala Cys
            180                 185                 190
His Pro Cys Ser Pro Met Cys Lys Gly Ser Arg Cys Trp Gly Glu Ser
        195                 200                 205
Ser Glu Asp Cys Gln Ser Leu Thr Arg Thr Val Cys Ala Gly Gly Cys
    210                 215                 220
Ala Arg Cys Lys Gly Pro Leu Pro Thr Asp Cys Cys His Glu Gln Cys
225                 230                 235                 240
Ala Ala Gly Cys Thr Gly Pro Lys His Ser Asp Cys Leu Ala Cys Leu
                245                 250                 255
His Phe Asn His Ser Gly Ile Cys Glu Leu His Cys Pro Ala Leu Val
            260                 265                 270
Thr Tyr Asn Thr Asp Thr Phe Glu Ser Met Pro Asn Pro Glu Gly Arg
        275                 280                 285
Tyr Thr Phe Gly Ala Ser Cys Val Thr Ala Cys Pro Tyr Asn Tyr Leu
    290                 295                 300
Ser Thr Asp Val Gly Ser Cys Thr Leu Val Cys Pro Leu His Asn Gln
305                 310                 315                 320
Glu Val Thr Ala Glu Asp Gly Thr Gln Arg Cys Glu Lys Cys Ser Lys
                325                 330                 335
Pro Cys Ala Arg Val Cys Tyr Gly Leu Gly Met Glu His Leu Arg Glu
            340                 345                 350
Val Arg Ala Val Thr Ser Ala Asn Ile Gln Glu Phe Ala Gly Cys Lys
        355                 360                 365
Lys Ile Phe Gly Ser Leu Ala Phe Leu Pro Glu Ser Phe Asp Gly Asp
    370                 375                 380
Pro Ala Ser Asn Thr Ala Pro Leu Gln Pro Glu Gln Leu Gln Val Phe
385                 390                 395                 400
Glu Thr Leu Glu Glu Ile Thr Gly Tyr Leu Tyr Ile Ser Ala Trp Pro
                405                 410                 415
Asp Ser Leu Pro Asp Leu Ser Val Phe Gln Asn Leu Gln Val Ile Arg
            420                 425                 430
Gly Arg Ile Leu His Asn Gly Ala Tyr Ser Leu Thr Leu Gln Gly Leu
        435                 440                 445
```

```
Gly Ile Ser Trp Leu Gly Leu Arg Ser Leu Arg Glu Leu Gly Ser Gly
    450                 455                 460
Leu Ala Leu Ile His His Asn Thr His Leu Cys Phe Val His Thr Val
465                 470                 475                 480
Pro Trp Asp Gln Leu Phe Arg Asn Pro His Gln Ala Leu Leu His Thr
                485                 490                 495
Ala Asn Arg Pro Glu Asp Glu Cys Val Gly Glu Gly Leu Ala Cys His
            500                 505                 510
Gln Leu Cys Ala Arg Gly His Cys Trp Gly Pro Gly Pro Thr Gln Cys
        515                 520                 525
Val Asn Cys Ser Gln Phe Leu Arg Gly Gln Glu Cys Val Glu Glu Cys
    530                 535                 540
Arg Val Leu Gln Gly Leu Pro Arg Glu Tyr Val Asn Ala Arg His Cys
545                 550                 555                 560
Leu Pro Cys His Pro Glu Cys Gln Pro Gln Asn Gly Ser Val Thr Cys
                565                 570                 575
Phe Gly Pro Glu Ala Asp Gln Cys Val Ala Cys Ala His Tyr Lys Asp
            580                 585                 590
Pro Pro Phe Cys Val Ala Arg Cys Pro Ser Gly Val Lys Pro Asp Leu
        595                 600                 605
Ser Tyr Met Pro Ile Trp Lys Phe Pro Asp Glu Glu Gly Ala Cys Gln
    610                 615                 620
Pro Cys Pro Ile Asn Cys Thr His Ser Cys Val Asp Leu Asp Asp Lys
625                 630                 635                 640
Gly Cys Pro Ala Glu Gln Arg Ala Ser Pro Leu Thr Ser Ile Ile Ser
                645                 650                 655
Ala Val Val Gly Ile Leu Leu Val Val Val Leu Gly Val Val Phe Gly
            660                 665                 670
Ile Leu Ile Lys Arg Arg Gln Gln Lys Ile Arg Lys Tyr Thr Met Arg
        675                 680                 685
Arg Leu Leu Gln Glu Thr Glu Leu Val Glu Pro Leu Thr Pro Ser Gly
    690                 695                 700
Ala Met Pro Asn Gln Ala Gln Met Arg Ile Leu Lys Glu Thr Glu Leu
705                 710                 715                 720
Arg Lys Val Lys Val Leu Gly Ser Gly Ala Phe Gly Thr Val Tyr Lys
                725                 730                 735
Gly Ile Trp Ile Pro Asp Gly Glu Asn Val Lys Ile Pro Val Ala Ile
            740                 745                 750
Lys Val Leu Arg Glu Asn Thr Ser Pro Lys Ala Asn Lys Glu Ile Leu
        755                 760                 765
Asp Glu Ala Tyr Val Met Ala Gly Val Gly Ser Pro Tyr Val Ser Arg
    770                 775                 780
Leu Leu Gly Ile Cys Leu Thr Ser Thr Val Gln Leu Val Thr Gln Leu
785                 790                 795                 800
Met Pro Tyr Gly Cys Leu Leu Asp His Val Arg Glu Asn Arg Gly Arg
                805                 810                 815
Leu Gly Ser Gln Asp Leu Leu Asn Trp Cys Met Gln Ile Ala Lys Gly
            820                 825                 830
Met Ser Tyr Leu Glu Asp Val Arg Leu Val His Arg Asp Leu Ala Ala
        835                 840                 845
Arg Asn Val Leu Val Lys Ser Pro Asn His Val Lys Ile Thr Asp Phe
    850                 855                 860
Gly Leu Ala Arg Leu Leu Asp Ile Asp Glu Thr Glu Tyr His Ala Asp
865                 870                 875                 880
```

```
Gly Gly Lys Val Pro Ile Lys Trp Met Ala Leu Glu Ser Ile Leu Arg
                885                 890                 895
Arg Arg Phe Thr His Gln Ser Asp Val Trp Ser Tyr Gly Val Thr Val
            900                 905                 910
Trp Glu Leu Met Thr Phe Gly Ala Lys Pro Tyr Asp Gly Ile Pro Ala
        915                 920                 925
Arg Glu Ile Pro Asp Leu Leu Glu Lys Gly Glu Arg Leu Pro Gln Pro
    930                 935                 940
Pro Ile Cys Thr Ile Asp Val Tyr Met Ile Met Val Lys Cys Trp Met
945                 950                 955                 960
Ile Asp Ser Glu Cys Arg Pro Arg Phe Arg Glu Leu Val Ser Glu Phe
                965                 970                 975
Ser Arg Met Ala Arg Asp Pro Gln Arg Phe Val Val Ile Gln Asn Glu
            980                 985                 990
Asp Leu Gly Pro Ala Ser Pro Leu Asp Ser Thr Phe Tyr Arg Ser Leu
        995                 1000                1005
Leu Glu Asp Asp Asp Met Gly Asp Leu Val Asp Ala Glu Glu Tyr Leu
    1010                1015                1020
Val Pro Gln Gln Gly Phe Phe Cys Pro Asp Pro Ala Pro Gly Ala Gly
1025                1030                1035                1040
Gly Met Val His His Arg His Arg Ser Ser Ser Thr Arg Ser Gly Gly
                1045                1050                1055
Gly Asp Leu Thr Leu Gly Leu Glu Pro Ser Glu Glu Glu Ala Pro Arg
            1060                1065                1070
Ser Pro Leu Ala Pro Ser Glu Gly Ala Gly Ser Asp Val Phe Asp Gly
        1075                1080                1085
Asp Leu Gly Met Gly Ala Ala Lys Gly Leu Gln Ser Leu Pro Thr His
    1090                1095                1100
Asp Pro Ser Pro Leu Gln Arg Tyr Ser Glu Asp Pro Thr Val Pro Leu
1105                1110                1115                1120
Pro Ser Glu Thr Asp Gly Tyr Val Ala Pro Leu Thr Cys Ser Pro Gln
                1125                1130                1135
Pro Glu Tyr Val Asn Gln Pro Asp Val Arg Pro Gln Pro Pro Ser Pro
            1140                1145                1150
Arg Glu Gly Pro Leu Pro Ala Ala Arg Pro Ala Gly Ala Thr Leu Glu
        1155                1160                1165
Arg Pro Lys Thr Leu Ser Pro Gly Lys Asn Gly Val Val Lys Asp Val
    1170                1175                1180
Phe Ala Phe Gly Gly Ala Val Glu Asn Pro Glu Tyr Leu Thr Pro Gln
1185                1190                1195                1200
Gly Gly Ala Ala Pro Gln Pro His Pro Pro Pro Ala Phe Ser Pro Ala
                1205                1210                1215
Phe Asp Asn Leu Tyr Tyr Trp Asp Gln Asp Pro Pro Glu Arg Gly Ala
            1220                1225                1230
Pro Pro Ser Thr Phe Lys Gly Thr Pro Thr Ala Glu Asn Pro Glu Tyr
        1235                1240                1245
Leu Gly Leu Asp Val Pro Val
    1250                1255
```

<210> 24
<211> 9
<212> PRT
<213> Artificial Sequence

<220>
<223> HLA-A2 anchor motif

<220>
<221> MOD_RES
<222> (3)...(8)
<223> Xaa = any amino acid

<400> 24
Phe Leu Xaa Xaa Xaa Xaa Xaa Xaa Val
1 5

<210> 25
<211> 875
<212> PRT
<213> Clostridium tetani

<220>
<223> tetanus toxin (tetX)

<400> 25
Lys Ile Ile Pro Pro Thr Asn Ile Arg Glu Asn Leu Tyr Asn Arg Thr
1 5 10 15
Ala Ser Leu Thr Asp Leu Gly Gly Glu Leu Cys Ile Lys Ile Lys Asn
20 25 30
Glu Asp Leu Thr Phe Ile Ala Glu Lys Asn Ser Phe Ser Glu Glu Pro
35 40 45
Phe Gln Asp Glu Ile Val Ser Tyr Asn Thr Lys Asn Lys Pro Leu Asn
50 55 60
Phe Asn Tyr Ser Leu Asp Lys Ile Ile Val Asp Tyr Asn Leu Gln Ser
65 70 75 80
Lys Ile Thr Leu Pro Asn Asp Arg Thr Thr Pro Val Thr Lys Gly Ile
85 90 95
Pro Tyr Ala Pro Glu Tyr Lys Ser Asn Ala Ala Ser Thr Ile Glu Ile
100 105 110
His Asn Ile Asp Asp Asn Thr Ile Tyr Gln Tyr Leu Tyr Ala Gln Lys
115 120 125
Ser Pro Thr Thr Leu Gln Arg Ile Thr Met Thr Asn Ser Val Asp Asp
130 135 140
Ala Leu Ile Asn Ser Thr Lys Ile Tyr Ser Tyr Phe Pro Ser Val Ile
145 150 155 160
Ser Lys Val Asn Gln Gly Ala Gln Gly Ile Leu Phe Leu Gln Trp Val
165 170 175
Arg Asp Ile Ile Asp Asp Phe Thr Asn Glu Ser Ser Gln Lys Thr Thr
180 185 190
Ile Asp Lys Ile Ser Asp Val Ser Thr Ile Val Pro Tyr Ile Gly Pro
195 200 205
Ala Leu Asn Ile Val Lys Gln Gly Tyr Glu Gly Asn Phe Ile Gly Ala
210 215 220
Leu Glu Thr Thr Gly Val Val Leu Leu Leu Glu Tyr Ile Pro Glu Ile
225 230 235 240
Thr Leu Pro Val Ile Ala Ala Leu Ser Ile Ala Glu Ser Ser Thr Gln

```
                245                 250                 255
Lys Glu Lys Ile Ile Lys Thr Ile Asp Asn Phe Leu Glu Lys Arg Tyr
            260                 265                 270
Glu Lys Trp Ile Glu Val Tyr Lys Leu Val Lys Ala Lys Trp Leu Gly
        275                 280                 285
Thr Val Asn Thr Gln Phe Gln Lys Arg Ser Tyr Gln Met Tyr Arg Ser
    290                 295                 300
Leu Glu Tyr Gln Val Asp Ala Ile Lys Lys Ile Ile Asp Tyr Glu Tyr
305                 310                 315                 320
Lys Ile Tyr Ser Gly Pro Asp Lys Glu Gln Ile Ala Asp Glu Ile Asn
                325                 330                 335
Asn Leu Lys Asn Lys Leu Glu Glu Lys Ala Asn Lys Ala Met Ile Asn
            340                 345                 350
Ile Asn Ile Phe Met Arg Glu Ser Ser Arg Ser Phe Leu Val Asn Gln
        355                 360                 365
Met Ile Asn Glu Ala Lys Lys Gln Leu Leu Glu Phe Asp Thr Gln Ser
    370                 375                 380
Lys Asn Ile Leu Met Gln Tyr Ile Lys Ala Asn Ser Lys Phe Ile Gly
385                 390                 395                 400
Ile Thr Glu Leu Lys Lys Leu Glu Ser Lys Ile Asn Lys Val Phe Ser
                405                 410                 415
Thr Pro Ile Pro Phe Ser Tyr Ser Lys Asn Leu Asp Cys Trp Val Asp
            420                 425                 430
Asn Glu Glu Asp Ile Asp Val Ile Leu Lys Lys Ser Thr Ile Leu Asn
        435                 440                 445
Leu Asp Ile Asn Asn Asp Ile Ile Ser Asp Ile Ser Gly Phe Asn Ser
    450                 455                 460
Ser Val Ile Thr Tyr Pro Asp Ala Gln Leu Val Pro Gly Ile Asn Gly
465                 470                 475                 480
Lys Ala Ile His Leu Val Asn Asn Glu Ser Ser Glu Val Ile Val His
                485                 490                 495
Lys Ala Met Asp Ile Glu Tyr Asn Asp Met Phe Asn Asn Phe Thr Val
            500                 505                 510
Ser Phe Trp Leu Arg Val Pro Lys Val Ser Ala Ser His Leu Glu Gln
        515                 520                 525
Tyr Gly Thr Asn Glu Tyr Ser Ile Ile Ser Ser Met Lys Lys His Ser
    530                 535                 540
Leu Ser Ile Gly Ser Gly Trp Ser Val Ser Leu Lys Gly Asn Asn Leu
545                 550                 555                 560
Ile Trp Thr Leu Lys Asp Ser Ala Gly Glu Val Arg Gln Ile Thr Phe
                565                 570                 575
Arg Asp Leu Pro Asp Lys Phe Asn Ala Tyr Leu Ala Asn Lys Trp Val
            580                 585                 590
Phe Ile Thr Ile Thr Asn Asp Arg Leu Ser Ser Ala Asn Leu Tyr Ile
        595                 600                 605
Asn Gly Val Leu Met Gly Ser Ala Glu Ile Thr Gly Leu Gly Ala Ile
    610                 615                 620
Arg Glu Asp Asn Asn Ile Thr Leu Lys Leu Asp Arg Cys Asn Asn Asn
625                 630                 635                 640
Asn Gln Tyr Val Ser Ile Asp Lys Phe Arg Ile Phe Cys Lys Ala Leu
                645                 650                 655
Asn Pro Lys Glu Ile Glu Lys Leu Tyr Thr Ser Tyr Leu Ser Ile Thr
            660                 665                 670
Phe Leu Arg Asp Phe Trp Gly Asn Pro Leu Arg Tyr Asp Thr Glu Tyr
```

```
        675                 680                 685
Tyr Leu Ile Pro Val Ala Ser Ser Ser Lys Asp Val Gln Leu Lys Asn
    690                 695                 700
Ile Thr Asp Tyr Met Tyr Leu Thr Asn Ala Pro Ser Tyr Thr Asn Gly
705                 710                 715                 720
Lys Leu Asn Ile Tyr Tyr Arg Arg Leu Tyr Asn Gly Leu Lys Phe Ile
                725                 730                 735
Ile Lys Arg Tyr Thr Pro Asn Asn Glu Ile Asp Ser Phe Val Lys Ser
            740                 745                 750
Gly Asp Phe Ile Lys Leu Tyr Val Ser Tyr Asn Asn Asn Glu His Ile
        755                 760                 765
Val Gly Tyr Pro Lys Asp Gly Asn Ala Phe Asn Asn Leu Asp Arg Ile
    770                 775                 780
Leu Arg Val Gly Tyr Asn Ala Pro Gly Ile Pro Leu Tyr Lys Lys Met
785                 790                 795                 800
Glu Ala Val Lys Leu Arg Asp Leu Lys Thr Tyr Ser Val Gln Leu Lys
                805                 810                 815
Leu Tyr Asp Asp Lys Asn Ala Ser Leu Gly Leu Val Gly Thr His Asn
            820                 825                 830
Gly Gln Ile Gly Asn Asp Pro Asn Arg Asp Ile Leu Ile Ala Ser Asn
        835                 840                 845
Trp Tyr Phe Asn His Leu Lys Asp Lys Ile Leu Gly Cys Asp Trp Tyr
    850                 855                 860
Phe Val Pro Thr Asp Glu Gly Trp Thr Asn Asp
865                 870                 875


<210> 26
<211> 15
<212> PRT
<213> Artificial Sequence

<220>
<223> universal T helper epitope

<400> 26
Gln Tyr Ile Lys Ala Asn Ser Lys Phe Ile Gly Ile Thr Glu Leu
 1               5                  10                  15


<210> 27
<211> 190
<212> PRT
<213> Human immunodeficiency virus type 1

<220>
<223> HIV-1 reverse transcriptase (pol protein)

<400> 27
Glu Ile Cys Thr Glu Met Glu Lys Glu Gly Lys Ile Ser Lys Ile Gly
 1               5                  10                  15
Pro Glu Asn Pro Tyr Asn Thr Pro Val Phe Ala Ile Lys Lys Lys Asp
            20                  25                  30
Ser Thr Lys Trp Arg Lys Leu Val Asp Phe Arg Glu Leu Asn Lys Arg
```

```
        35                  40                  45
Thr Gln Asp Phe Trp Glu Val Gln Leu Gly Ile Pro His Pro Ala Gly
    50                  55                  60
Leu Lys Lys Lys Lys Ser Val Thr Val Leu Asp Val Gly Asp Ala Tyr
65                  70                  75                  80
Phe Ser Val Pro Leu Asp Lys Asp Phe Arg Lys Tyr Thr Ala Phe Thr
                85                  90                  95
Ile Pro Ser Thr Asn Asn Glu Thr Pro Gly Ile Arg Tyr Gln Tyr Asn
            100                 105                 110
Val Leu Pro Gln Gly Trp Lys Gly Ser Pro Ala Ile Phe Gln Ser Ser
        115                 120                 125
Met Thr Lys Ile Leu Glu Pro Phe Arg Lys Gln Asn Pro Glu Ile Val
    130                 135                 140
Ile Tyr Gln Tyr Met Asp Asp Leu Tyr Ile Gly Ser Asp Leu Glu Ile
145                 150                 155                 160
Gly Gln His Arg Thr Lys Ile Glu Glu Leu Arg Gln His Leu Leu Lys
                165                 170                 175
Trp Gly Leu Thr Thr Pro Asp Lys Lys His Gln Lys Glu Pro
            180                 185                 190
```

<210> 28
<211> 20
<212> PRT
<213> Artificial Sequence

<220>
<223> universal T helper epitope

<400> 28
```
Phe Arg Lys Gln Asn Pro Asp Ile Val Ile Tyr Gln Tyr Met Asp Asp
 1               5                  10                  15
Leu Tyr Val Gly
            20
```

<210> 29
<211> 236
<212> PRT
<213> Human immunodeficiency virus type 1

<220>
<223> HIV-1 gag protein

<400> 29
```
Trp Asp Arg Leu His Pro Ala Gln Ala Gly Pro Ile Ala Pro Gly Gln
 1               5                  10                  15
Ile Arg Glu Pro Arg Gly Ser Asp Ile Ala Gly Thr Thr Ser Thr Leu
            20                  25                  30
Gln Glu Gln Ile Thr Trp Met Thr Asn Asn Pro Pro Ile Pro Val Gly
        35                  40                  45
Glu Ile Tyr Lys Arg Trp Ile Ile Leu Gly Leu Asn Lys Ile Val Arg
    50                  55                  60
Met Tyr Ser Pro Val Ser Ile Leu Asp Ile Lys Gln Gly Pro Lys Glu
```

```
65                  70                  75                  80
Pro Phe Arg Asp Tyr Val Asp Arg Phe Phe Lys Ala Leu Arg Ala Glu
                85                  90                  95
Gln Ala Thr Gln Asp Val Lys Asn Trp Met Thr Asp Thr Leu Leu Val
            100                 105                 110
Gln Asn Ala Asn Pro Asp Cys Lys Ser Ile Leu Arg Gly Leu Gly Pro
        115                 120                 125
Gly Ala Ser Leu Glu Glu Met Met Thr Ala Cys Gln Gly Val Gly Gly
    130                 135                 140
Pro Ser His Lys Ala Arg Val Leu Ala Glu Ala Met Ser Gln Ala Asn
145                 150                 155                 160
Ser Val Asn Met Met Gln Arg Ser Asn Phe Lys Gly Pro Lys Arg Thr
                165                 170                 175
Val Lys Cys Phe Asn Cys Gly Lys Glu Gly His Ile Ala Arg Asn Cys
            180                 185                 190
Arg Ala Pro Arg Lys Lys Gly Cys Trp Lys Cys Gly Gln Glu Gly His
        195                 200                 205
Gln Met Lys Asp Cys Thr Glu Arg Gln Ala Asn Phe Leu Gly Lys Ile
    210                 215                 220
Trp Pro Ser His Lys Gly Arg Pro Gly Asn Phe Leu
225                 230                 235


<210> 30
<211> 110
<212> PRT
<213> Homo sapiens

<220>
<223> preproinsulin, insulin (INS) precursor

<400> 30
Met Ala Leu Trp Met Arg Leu Leu Pro Leu Leu Ala Leu Leu Ala Leu
 1               5                  10                  15
Trp Gly Pro Asp Pro Ala Ala Ala Phe Val Asn Gln His Leu Cys Gly
            20                  25                  30
Ser His Leu Val Glu Ala Leu Tyr Leu Val Cys Gly Glu Arg Gly Phe
        35                  40                  45
Phe Tyr Thr Pro Lys Thr Arg Arg Glu Ala Glu Asp Leu Gln Val Gly
    50                  55                  60
Gln Val Glu Leu Gly Gly Gly Pro Gly Ala Gly Ser Leu Gln Pro Leu
65                  70                  75                  80
Ala Leu Glu Gly Ser Leu Gln Lys Arg Gly Ile Val Glu Gln Cys Cys
                85                  90                  95
Thr Ser Ile Cys Ser Leu Tyr Gln Leu Glu Asn Tyr Cys Asn
            100                 105                 110


<210> 31
<211> 585
<212> PRT
<213> Homo sapiens

<220>
```

<223> glutamic acid decarboxylase 2 (GAD2), 65kDa isoform (GAD65)

<400> 31

```
Met Ala Ser Pro Gly Ser Gly Phe Trp Ser Phe Gly Ser Glu Asp Gly
 1               5                  10                  15
Ser Gly Asp Ser Glu Asn Pro Gly Thr Ala Arg Ala Trp Cys Gln Val
            20                  25                  30
Ala Gln Lys Phe Thr Gly Gly Ile Gly Asn Lys Leu Cys Ala Leu Leu
        35                  40                  45
Tyr Gly Asp Ala Glu Lys Pro Ala Glu Ser Gly Gly Ser Gln Pro Pro
    50                  55                  60
Arg Ala Ala Ala Arg Lys Ala Ala Cys Ala Cys Asp Gln Lys Pro Cys
65                  70                  75                  80
Ser Cys Ser Lys Val Asp Val Asn Tyr Ala Phe Leu His Ala Thr Asp
                85                  90                  95
Leu Leu Pro Ala Cys Asp Gly Glu Arg Pro Thr Leu Ala Phe Leu Gln
            100                 105                 110
Asp Val Met Asn Ile Leu Leu Gln Tyr Val Val Lys Ser Phe Asp Arg
        115                 120                 125
Ser Thr Lys Val Ile Asp Phe His Tyr Pro Asn Glu Leu Leu Gln Glu
    130                 135                 140
Tyr Asn Trp Glu Leu Ala Asp Gln Pro Gln Asn Leu Glu Glu Ile Leu
145                 150                 155                 160
Met His Cys Gln Thr Thr Leu Lys Tyr Ala Ile Lys Thr Gly His Pro
                165                 170                 175
Arg Tyr Phe Asn Gln Leu Ser Thr Gly Leu Asp Met Val Gly Leu Ala
            180                 185                 190
Ala Asp Trp Leu Thr Ser Thr Ala Asn Thr Asn Met Phe Thr Tyr Glu
        195                 200                 205
Ile Ala Pro Val Phe Val Leu Leu Glu Tyr Val Thr Leu Lys Lys Met
    210                 215                 220
Arg Glu Ile Ile Gly Trp Pro Gly Gly Ser Gly Asp Gly Ile Phe Ser
225                 230                 235                 240
Pro Gly Gly Ala Ile Ser Asn Met Tyr Ala Met Met Ile Ala Arg Phe
                245                 250                 255
Lys Met Phe Pro Glu Val Lys Glu Lys Gly Met Ala Ala Leu Pro Arg
            260                 265                 270
Leu Ile Ala Phe Thr Ser Glu His Ser His Phe Ser Leu Lys Lys Gly
        275                 280                 285
Ala Ala Ala Leu Gly Ile Gly Thr Asp Ser Val Ile Leu Ile Lys Cys
    290                 295                 300
Asp Glu Arg Gly Lys Met Ile Pro Ser Asp Leu Glu Arg Arg Ile Leu
305                 310                 315                 320
Glu Ala Lys Gln Lys Gly Phe Val Pro Phe Leu Val Ser Ala Thr Ala
                325                 330                 335
Gly Thr Thr Val Tyr Gly Ala Phe Asp Pro Leu Leu Ala Val Ala Asp
            340                 345                 350
Ile Cys Lys Lys Tyr Lys Ile Trp Met His Val Asp Ala Ala Trp Gly
        355                 360                 365
Gly Gly Leu Leu Met Ser Arg Lys His Lys Trp Lys Leu Ser Gly Val
    370                 375                 380
Glu Arg Ala Asn Ser Val Thr Trp Asn Pro His Lys Met Met Gly Val
385                 390                 395                 400
```

```
Pro Leu Gln Cys Ser Ala Leu Leu Val Arg Glu Glu Gly Leu Met Gln
                405                 410                 415
Asn Cys Asn Gln Met His Ala Ser Tyr Leu Phe Gln Gln Asp Lys His
            420                 425                 430
Tyr Asp Leu Ser Tyr Asp Thr Gly Asp Lys Ala Leu Gln Cys Gly Arg
        435                 440                 445
His Val Asp Val Phe Lys Leu Trp Leu Met Trp Arg Ala Lys Gly Thr
    450                 455                 460
Thr Gly Phe Glu Ala His Val Asp Lys Cys Leu Glu Leu Ala Glu Tyr
465                 470                 475                 480
Leu Tyr Asn Ile Ile Lys Asn Arg Glu Gly Tyr Glu Met Val Phe Asp
                485                 490                 495
Gly Lys Pro Gln His Thr Asn Val Cys Phe Trp Tyr Ile Pro Pro Ser
            500                 505                 510
Leu Arg Thr Leu Glu Asp Asn Glu Glu Arg Met Ser Arg Leu Ser Lys
        515                 520                 525
Val Ala Pro Val Ile Lys Ala Arg Met Met Glu Tyr Gly Thr Thr Met
    530                 535                 540
Val Ser Tyr Gln Pro Leu Gly Asp Lys Val Asn Phe Phe Arg Met Val
545                 550                 555                 560
Ile Ser Asn Pro Ala Ala Thr His Gln Asp Ile Asp Phe Leu Ile Glu
                565                 570                 575
Glu Ile Glu Arg Leu Gly Gln Asp Leu
            580                 585


<210> 32
<211> 74
<212> PRT
<213> Artificial Sequence

<220>
<223> composite self epitope Insulin-GAD

<220>
<221> MOD_RES
<222> (1)...(74)
<223> Xaa = any amino acid

<400> 32
Cys Gly Ser His Leu Val Glu Ala Leu Tyr Leu Val Cys Gly Glu Arg
 1               5                  10                  15
Gly Xaa Xaa Xaa Xaa Pro Arg Leu Ile Ala Phe Thr Ser Glu His Ser
            20                  25                  30
His Phe Ser Leu Xaa Xaa Xaa Xaa Leu Tyr Asn Ile Ile Lys Asn Arg
        35                  40                  45
Glu Gly Tyr Glu Met Val Phe Xaa Xaa Xaa Xaa Pro Ser Leu Arg Thr
    50                  55                  60
Leu Glu Asp Asn Glu Glu Arg Met Ser Arg
65                  70


<210> 33
<211> 54
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<212> PRT
<213> Artificial Sequence

<220>
<223> composite non-self epitope Tetanus-gp100, MART-1,
TRP-2

<220>
<221> MOD_RES
<222> (1)...(54)
<223> Xaa = any amino acid

<400> 33
Gln Tyr Ile Lys Ala Asn Ser Lys Phe Ile Gly Ile Thr Glu Leu Xaa
1 5 10 15
Xaa Xaa Xaa Phe Leu Asp Gln Val Ala Phe Ser Val Xaa Xaa Xaa Xaa
20 25 30
Ala Ala Gly Ile Gly Ile Leu Thr Val Xaa Xaa Xaa Xaa Ser Val Arg
35 40 45
Asp Thr Leu Leu Gly Gly
50

<210> 34
<211> 146
<212> PRT
<213> Homo sapiens

<220>
<223> prostate associated gene 1 (PAGE-1), P antigen
family, member 1 (prostate associated), isoform
CRA_b, G antigen family B, member 1 (GAGEB1,
GAGE9)

<400> 34
Met Gly Phe Leu Arg Arg Leu Ile Tyr Arg Arg Arg Pro Met Ile Tyr
1 5 10 15
Val Glu Ser Ser Glu Glu Ser Ser Asp Glu Gln Pro Asp Glu Val Glu
20 25 30
Ser Pro Thr Gln Ser Gln Asp Ser Thr Pro Ala Glu Glu Arg Glu Asp
35 40 45
Glu Gly Ala Ser Ala Ala Gln Gly Gln Glu Pro Glu Ala Asp Ser Gln
50 55 60
Glu Leu Val Gln Pro Lys Thr Gly Cys Glu Pro Gly Asp Gly Pro Asp
65 70 75 80
Thr Lys Arg Val Cys Leu Arg Asn Glu Glu Gln Met Lys Leu Pro Ala
85 90 95
Glu Gly Pro Glu Pro Glu Ala Asp Ser Gln Glu Gln Val His Pro Lys
100 105 110
Thr Gly Cys Glu Arg Gly Asp Gly Pro Asp Val Gln Glu Leu Gly Leu
115 120 125
Pro Asn Pro Glu Glu Val Lys Thr Pro Glu Glu Asp Glu Gly Gln Ser
130 135 140
Gln Pro

145

<210> 35
<211> 339
<212> PRT
<213> Homo sapiens

<220>
<223> six transmembrane epithelial antigen of the prostate (STEAP1), metalloreductase STEAP1, six transmembrane protein of the prostate 1 (STAMP1)

<400> 35

```
Met Glu Ser Arg Lys Asp Ile Thr Asn Gln Glu Glu Leu Trp Lys Met
 1               5                  10                  15
Lys Pro Arg Arg Asn Leu Glu Glu Asp Asp Tyr Leu His Lys Asp Thr
            20                  25                  30
Gly Glu Thr Ser Met Leu Lys Arg Pro Val Leu Leu His Leu His Gln
        35                  40                  45
Thr Ala His Ala Asp Glu Phe Asp Cys Pro Ser Glu Leu Gln His Thr
    50                  55                  60
Gln Glu Leu Phe Pro Gln Trp His Leu Pro Ile Lys Ile Ala Ala Ile
65                  70                  75                  80
Ile Ala Ser Leu Thr Phe Leu Tyr Thr Leu Leu Arg Glu Val Ile His
                85                  90                  95
Pro Leu Ala Thr Ser His Gln Gln Tyr Phe Tyr Lys Ile Pro Ile Leu
            100                 105                 110
Val Ile Asn Lys Val Leu Pro Met Val Ser Ile Thr Leu Leu Ala Leu
        115                 120                 125
Val Tyr Leu Pro Gly Val Ile Ala Ala Ile Val Gln Leu His Asn Gly
    130                 135                 140
Thr Lys Tyr Lys Lys Phe Pro His Trp Leu Asp Lys Trp Met Leu Thr
145                 150                 155                 160
Arg Lys Gln Phe Gly Leu Leu Ser Phe Phe Phe Ala Val Leu His Ala
                165                 170                 175
Ile Tyr Ser Leu Ser Tyr Pro Met Arg Arg Ser Tyr Arg Tyr Lys Leu
            180                 185                 190
Leu Asn Trp Ala Tyr Gln Gln Val Gln Gln Asn Lys Glu Asp Ala Trp
        195                 200                 205
Ile Glu His Asp Val Trp Arg Met Glu Ile Tyr Val Ser Leu Gly Ile
    210                 215                 220
Val Gly Leu Ala Ile Leu Ala Leu Leu Ala Val Thr Ser Ile Pro Ser
225                 230                 235                 240
Val Ser Asp Ser Leu Thr Trp Arg Glu Phe His Tyr Ile Gln Ser Lys
                245                 250                 255
Leu Gly Ile Val Ser Leu Leu Leu Gly Thr Ile His Ala Leu Ile Phe
            260                 265                 270
Ala Trp Asn Lys Trp Ile Asp Ile Lys Gln Phe Val Trp Tyr Thr Pro
        275                 280                 285
Pro Thr Phe Met Ile Ala Val Phe Leu Pro Ile Val Val Leu Ile Phe
    290                 295                 300
Lys Ser Ile Leu Phe Leu Pro Cys Leu Arg Lys Lys Ile Leu Lys Ile
305                 310                 315                 320
```

```
Arg His Gly Trp Glu Asp Val Thr Lys Ile Asn Lys Thr Glu Ile Cys
                325                 330                 335
Ser Gln Leu
```

<210> 36
<211> 102
<212> PRT
<213> Homo sapiens

<220>
<223> G antigen, family C, member 1, prostate-associated gene protein 4 (PAGE4), JM27 protein

<400> 36

```
Met Ser Ala Arg Val Arg Ser Arg Ser Arg Gly Arg Gly Asp Gly Gln
 1               5                  10                  15
Glu Ala Pro Asp Val Val Ala Phe Val Ala Pro Gly Glu Ser Gln Gln
            20                  25                  30
Glu Glu Pro Pro Thr Asp Asn Gln Asp Ile Glu Pro Gly Gln Glu Arg
        35                  40                  45
Glu Gly Thr Pro Pro Ile Glu Glu Arg Lys Val Glu Gly Asp Cys Gln
    50                  55                  60
Glu Met Asp Leu Glu Lys Thr Arg Ser Glu Arg Gly Asp Gly Ser Asp
65                  70                  75                  80
Val Lys Glu Lys Thr Pro Pro Asn Pro Lys His Ala Lys Thr Lys Glu
                85                  90                  95
Ala Gly Asp Gly Gln Pro
            100
```

<210> 37
<211> 273
<212> PRT
<213> Homo sapiens

<220>
<223> mucin 1, transmembrane, isoform 1 precursor (MUC1), peanut-reactive urinary mucin, episialin, polymorphic epithelial mucin, tumor associated epithelial mucin, breast carcinoma-associated antigen DF3, H23 antigen

<400> 37

```
Met Thr Pro Gly Thr Gln Ser Pro Phe Phe Leu Leu Leu Leu Leu Thr
 1               5                  10                  15
Val Leu Thr Val Val Thr Gly Ser Gly His Ala Ser Ser Thr Pro Gly
            20                  25                  30
Gly Glu Lys Glu Thr Ser Ala Thr Gln Arg Ser Ser Val Pro Ser Ser
        35                  40                  45
Thr Glu Lys Asn Ala Leu Ser Thr Gly Val Ser Phe Phe Phe Leu Ser
    50                  55                  60
Phe His Ile Ser Asn Leu Gln Phe Asn Ser Ser Leu Glu Asp Pro Ser
65                  70                  75                  80
Thr Asp Tyr Tyr Gln Glu Leu Gln Arg Asp Ile Ser Glu Met Phe Leu
```

```
                85                  90                  95
Gln Ile Tyr Lys Gln Gly Gly Phe Leu Gly Leu Ser Asn Ile Lys Phe
            100                 105                 110
Arg Pro Gly Ser Val Val Val Gln Leu Thr Leu Ala Phe Arg Glu Gly
        115                 120                 125
Thr Ile Asn Val His Asp Val Glu Thr Gln Phe Asn Gln Tyr Lys Thr
    130                 135                 140
Glu Ala Ala Ser Arg Tyr Asn Leu Thr Ile Ser Asp Val Ser Val Ser
145                 150                 155                 160
Asp Val Pro Phe Pro Phe Ser Ala Gln Ser Gly Ala Gly Val Pro Gly
                165                 170                 175
Trp Gly Ile Ala Leu Leu Val Leu Val Cys Val Leu Val Ala Leu Ala
            180                 185                 190
Ile Val Tyr Leu Ile Ala Leu Ala Val Cys Gln Cys Arg Arg Lys Asn
        195                 200                 205
Tyr Gly Gln Leu Asp Ile Phe Pro Ala Arg Asp Thr Tyr His Pro Met
    210                 215                 220
Ser Glu Tyr Pro Thr Tyr His Thr His Gly Arg Tyr Val Pro Pro Ser
225                 230                 235                 240
Ser Thr Asp Arg Ser Pro Tyr Glu Lys Val Ser Ala Gly Asn Gly Gly
                245                 250                 255
Ser Ser Leu Ser Tyr Thr Asn Pro Ala Val Ala Ala Thr Ser Ala Asn
            260                 265                 270
Leu
```

<210> 38
<211> 263
<212> PRT
<213> Ovine respiratory syncytial virus

<220>
<223> Ovine RSV (ORSV) major surface glycoprotein G, attachment glycoprotein G, G glycoprotein

<400> 38

```
Met Ser Asn His Thr His His Phe Glu Phe Lys Thr Leu Lys Lys Ala
 1               5                  10                  15
Trp Lys Ala Ser Lys Tyr Phe Ile Val Gly Leu Ser Cys Leu Tyr Lys
            20                  25                  30
Leu Asn Leu Lys Ser Leu Val Gln Met Ala Leu Ser Ala Leu Ala Met
        35                  40                  45
Ile Thr Leu Val Ser Leu Thr Ile Thr Ala Ile Ile Tyr Ile Ser Thr
    50                  55                  60
Gly Asn Thr Lys Ala Lys Pro Met Pro Thr Pro Thr Ile Gln Ile Thr
65                  70                  75                  80
Gln Gln Phe Gln Asn His Thr Ser Leu Pro Pro Thr Glu His Asn His
                85                  90                  95
Asn Ser Thr His Ser Pro Thr Gln Gly Thr Thr Ser Pro His Thr Phe
            100                 105                 110
Ala Val Asp Val Thr Glu Gly Thr Arg Tyr Tyr His Leu Thr Leu Lys
        115                 120                 125
Thr Gln Gly Gly Lys Thr Lys Gly Pro Pro Thr Pro His Ala Thr Arg
    130                 135                 140
```

```
Lys Pro Pro Ile Ser Ser Gln Lys Ser Asn Pro Ser Glu Ile Gln Gln
145                 150                 155                 160
Asp Tyr Ser Asp Phe Gln Ile Leu Pro Tyr Val Pro Cys Asn Ile Cys
                165                 170                 175
Glu Gly Asp Ser Ala Cys Leu Ser Leu Cys Gln Asp Arg Ser Glu Ser
            180                 185                 190
Ile Leu Asp Lys Ala Leu Thr Thr Thr Pro Lys Lys Thr Pro Lys Pro
        195                 200                 205
Met Thr Thr Lys Lys Pro Thr Lys Thr Ser Thr His His Arg Thr Ser
    210                 215                 220
Leu Arg Asn Lys Leu Tyr Ile Lys Thr Asn Met Thr Thr Pro Pro His
225                 230                 235                 240
Gly Leu Ile Ser Thr Ala Lys His Asn Lys Asn Gln Ser Thr Val Gln
                245                 250                 255
Asn Pro Arg His Thr Leu Ala
            260
```

<210> 39
<211> 192
<212> PRT
<213> Human respiratory syncytial virus

<220>
<223> HRSV attachment G glycoprotein

<400> 39

```
Ala Thr Asp Gln Ile Lys Asn Thr Thr Pro Thr Tyr Leu Thr Gln Asn
 1               5                  10                  15
Pro Gln Leu Gly Ile Ser Phe Ser Asn Leu Ser Glu Thr Thr Ser Gln
            20                  25                  30
Pro Thr Thr Ile Leu Ala Ser Thr Thr Pro Ser Ala Glu Ser Thr Pro
        35                  40                  45
Gln Ser Thr Thr Val Lys Ile Lys Asn Thr Thr Thr Thr Gln Ile Gln
    50                  55                  60
Pro Ser Lys Pro Thr Thr Lys Gln Arg Gln Asn Lys Pro Gln Asn Lys
65                  70                  75                  80
Pro Asn Asn Asp Phe His Phe Glu Val Phe Asn Phe Val Pro Cys Ser
                85                  90                  95
Ile Cys Ser Asn Asn Pro Thr Cys Trp Ala Ile Cys Lys Arg Ile Pro
            100                 105                 110
Asn Lys Lys Pro Gly Lys Lys Thr Thr Thr Lys Pro Thr Lys Lys Pro
        115                 120                 125
Thr Ile Lys Thr Thr Lys Lys Asp Pro Lys Pro Gln Thr Thr Lys Pro
    130                 135                 140
Lys Glu Val Leu Thr Thr Lys Pro Thr Glu Lys Pro Thr Ile Ser Thr
145                 150                 155                 160
Thr Lys Thr Asn Ile Arg Thr Thr Leu Leu Thr Ser Asn Thr Thr Gly
                165                 170                 175
Asn Pro Glu His Thr Ser Gln Lys Gly Asn Pro Pro Leu Asn His Leu
            180                 185                 190
```

<210> 40

<211> 94
<212> PRT
<213> Human herpesvirus 1

<220>
<223> Herpes simplex virus type 1 (HSV-1) glycoprotein G, US4

<400> 40
Thr Pro Pro Met Pro Ser Ile Gly Leu Glu Glu Glu Glu Glu Glu Glu
1 5 10 15
Gly Ala Gly Asp Gly Glu His Leu Glu Gly Gly Asp Gly Thr Arg Asp
20 25 30
Thr Leu Pro Gln Ser Pro Gly Pro Ala Phe Pro Leu Ala Glu Asp Val
35 40 45
Glu Lys Asp Lys Pro Asn Arg Pro Val Val Pro Ser Pro Asp Pro Asn
50 55 60
Asn Ser Pro Ala Arg Pro Glu Thr Ser Arg Pro Lys Thr Pro Pro Thr
65 70 75 80
Ile Ile Gly Pro Leu Ala Thr Arg Pro Thr Thr Arg Leu Thr
85 90

<210> 41
<211> 14
<212> PRT
<213> Human herpesvirus 1

<220>
<223> Herpes simplex virus type 1 (HSV-1) immediate early protein infected cell protein 47 (ICP47), immediate-early protein IE12, Vmw12, immediate-early-5, US12, TAP transporter inhibitor ICP47

<400> 41
Met Ser Trp Ala Leu Glu Met Ala Asp Thr Phe Leu Asp Thr
1 5 10

<210> 42
<211> 261
<212> PRT
<213> Human herpesvirus 2

<220>
<223> Herpes simplex virus type 2 (HSV-2) neurovirulence factor infected cell protein 34.5 (ICP34.5), RL1 ORF

<400> 42
Met Ser Arg Arg Arg Gly Pro Arg Arg Arg Gly Pro Arg Arg Arg Pro
1 5 10 15
Arg Pro Gly Ala Pro Ala Val Pro Arg Pro Gly Ala Pro Ala Val Pro
20 25 30

```
Arg Pro Gly Ala Leu Pro Thr Ala Asp Ser Gln Met Val Pro Ala Tyr
        35                  40                  45
Asp Ser Gly Thr Ala Val Glu Ser Ala Pro Ala Ala Ser Ser Leu Leu
    50                  55                  60
Arg Arg Trp Leu Leu Val Pro Gln Ala Asp Asp Ser Asp Asp Ala Asp
65                  70                  75                  80
Tyr Ala Gly Asn Asp Asp Ala Glu Trp Ala Asn Ser Pro Pro Ser Glu
                85                  90                  95
Gly Gly Gly Lys Ala Pro Glu Ala Pro His Ala Ala Pro Ala Ala Ala
            100                 105                 110
Cys Pro Pro Pro Pro Pro Arg Lys Glu Arg Gly Pro Gln Arg Pro Leu
        115                 120                 125
Pro Pro His Leu Ala Leu Arg Leu Arg Thr Thr Thr Glu Tyr Leu Ala
    130                 135                 140
Arg Leu Ser Leu Arg Arg Arg Arg Pro Pro Ala Ser Pro Pro Ala Asp
145                 150                 155                 160
Ala Pro Arg Gly Lys Val Cys Phe Ser Pro Arg Val Gln Val Arg His
                165                 170                 175
Leu Val Ala Trp Glu Thr Ala Ala Arg Leu Ala Arg Arg Gly Ser Trp
            180                 185                 190
Ala Arg Glu Arg Ala Asp Arg Asp Arg Phe Arg Arg Arg Val Ala Ala
        195                 200                 205
Ala Glu Ala Val Ile Gly Pro Cys Leu Glu Pro Glu Ala Arg Ala Arg
    210                 215                 220
Ala Arg Ala Arg Ala Arg Ala His Glu Asp Gly Gly Pro Ala Glu Glu
225                 230                 235                 240
Glu Glu Ala Ala Ala Ala Ala Arg Gly Ser Ser Ala Ala Ala Gly Pro
                245                 250                 255
Gly Arg Arg Ala Val
            260
```

<210> 43
<211> 9
<212> PRT
<213> Artificial Sequence

<220>
<223> melanocyte lineage-specific antigen gp100 second epitope for agonists

<400> 43
```
Phe Leu Asp Gln Val Ala Phe Ser Val
 1               5
```

<210> 44
<211> 9
<212> PRT
<213> Artificial Sequence

<220>
<223> melanocyte lineage-specific antigen gp100 second epitope for agonists

<400> 44
Phe Leu Asp Gln Arg Val Phe Val Val
1 5

<210> 45
<211> 9
<212> PRT
<213> Artificial Sequence

<220>
<223> melanocyte lineage-specific antigen gp100 second epitope for agonists

<400> 45
Phe Leu Phe Leu Trp Phe Phe Glu Val
1 5

<210> 46
<211> 9
<212> PRT
<213> Artificial Sequence

<220>
<223> tyrosinase-related protein-2 (TRP-2) alternate epitope

<400> 46
Leu Leu Pro Gly Gly Arg Pro Tyr Arg
1 5

<210> 47
<211> 9
<212> PRT
<213> Artificial Sequence

<220>
<223> tyrosinase-related protein-2 (TRP-2) alternate epitope

<400> 47
Ser Val Tyr Asp Phe Phe Val Trp Leu
1 5

<210> 48
<211> 24
<212> PRT
<213> Artificial Sequence

<220>

<223> human IgG1 backbone Leader and Flanking sequence

<220>
<221> PEPTIDE
<222> (1)...(19)
<223> Leader sequence

<220>
<221> PEPTIDE
<222> (20)...(24)
<223> Flanking sequence

<400> 48
Met Lys His Leu Trp Phe Phe Leu Leu Leu Val Ala Ala Pro Arg Trp
 1   5   10   15
Val Leu Ser Gln Val Gln Leu Gln
            20

<210> 49
<211> 5
<212> PRT
<213> Artificial Sequence

<220>
<223> Poly-Gly Linker

<400> 49
Gly Gly Gly Gly Gly
 1   5

<210> 50
<211> 8
<212> PRT
<213> Artificial Sequence

<220>
<223> MHC class I-restricted OVA antigen peptide

<400> 50
Ser Ile Ile Asn Phe Glu Lys Leu
 1   5